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16. ABSTRACT

A review of the 1981 world literature was conducted on the effects of pollution on freshwater fish. This review contained 376 references and included papers on water quality such as dissolved gases and pH; chemical pollutants such as insecticides, herbicides, piscicides and anesthetics; inorganics, organics and industrial and municipal effluents including chlorine and wastewater and pulp and paper. Papers from reviews and symposia and those dealing with physical factors were also included. Within the above sections, papers were organized into general articles, surveys, toxicology, bioaccumulation, physiology and behavior.

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Effects of pollution on freshwater fish

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REVIEWS AND SYMPOSIA

Summaries of the Water Quality Criteria Documents for 64 priority toxic pollutants were published by the U. S. Environmental Protection Agency and included both aquatic-based and health-based criteria for pollutants or pollutant categories.[1] Supplementary information was given on the background for producing the documents, development of water quality criteria, relationships to regulatory programs, water quality standards, interpretation of criteria, and a summary of guidelines for deriving proposed criteria. Criteria for aquatic-life toxicology were based on acute and chronic toxicity to both freshwater and saltwater fish and to other aquatic organisms, including plants, and on residue data obtained for these organisms.

Spehar *et al.*[2] reviewed the effects of pollution on freshwater fish, including information on water quality, physical and chemical pollutants, and industrial and municipal effluents. A symposium proceedings concerning the aquatic toxicity of various pollutants, including Cu, CN, V, and chlorophenol, was published by the Fisheries and Marine Service (Canada).[3] The geochemical and biological processes involving heavy metal pollutants in contaminated rivers, lakes, and dams were reviewed by Foerstner and Prosi.[4] Emphasis was placed on recent developments in the study of toxic metal species and their availability to aquatic organisms, including fish. Examples were given to demonstrate the usefulness of sediment analyses to assess the hazards of metal pollutants to freshwater ecosystems and on the influence of abiotic factors on toxicity and uptake of heavy metals in fish. An annotated bibliography on biological effects of metals in the aquatic environment for the years 1975–1978 was produced by Eisler *et al.*[5] Aquatic species, such as fish, molluscs, crustaceans, and insects, were included in papers on toxicity, bioaccumulation, and physiological effects. The effects of Hg on fish and other aquatic life were reviewed by Taylor.[6] It was concluded that sublethal effects appeared at concentrations above 3 μg Hg^{++}/l of inorganic Hg and at levels above 0.1 μg Hg^{++}/l for organic Hg. Algae and larval stages of some molluscs and crustaceans appeared to be most sensitive to Hg. Thomas and Mathen[7] reviewed the position of Hg as a contaminant of fish and fishery products in India. The sources of Hg, its transformation in the environment, accumulation by fish, and its effects on human health were reviewed. Wilber[8] reviewed the aspects of Se toxicity and presented a complete discussion of the sources of Se and its biological effects on both freshwater and saltwater organisms. Bioconcentration factors (BCF) of 2 600 to 3 300 in fish were reported and levels of 60 μg Se/g of body tissue were found.

Mulla *et al.*[9] reviewed and analyzed literature on the impact of biological and chemical mosquito control agents on nontarget organisms in aquatic ecosystems. Larvivorous and predaceous fish were the more commonly utilized biological control agents. Most of the chemical control agents produced adverse effects on some nontarget organisms. Some chemical control agents were innocuous whereas others posed a great potential hazard to nontarget organisms. The pharmacology, toxicology, and degradation of endosulfan was reviewed by Gupta and Gupta.[10] Information on nomenclature, chemistry, metabolism by and toxicity to mammals, birds, fishes, and insects was summarized. Information on the degradation of endosulfan in water and plants and its metabolism in plants was included. A symposium on pesticide and xenobiotic metabolism in aquatic organisms, including fish, was held by the American Chemical Society.[11] The symposium consisted of papers on deposition, metabolism, biotransformation, fate, and degradation of several organic chemicals.

A review of literature concerning the relationship between biotransformation and toxicity of xenobiotic chemicals in fish[12] indicated that much of the metabolism of such chemicals was very similar in fish and mammals. Malyarevskaya[13] summarized 820 references on investigations of metabolism in fish exposed to natural and synthetic toxicants. Topics included characteristics of toxicants, bioaccumulation in fish, effects on blue-green algae, algal toxins, effects of pesticides on biochemical characteristics of fish, and metabolic effects of intoxication. Ahlborg and Thunberg[14] reviewed hazards involved in use of important chlorinated phenols with regard to toxicity of parent compounds and their contaminants, and as well as their degradation in the environment. The production and usage, occurrence and sources, acute and chronic toxicity, and mode of action were reviewed. A section on metabolism and toxicity of chlorinated phenols in fish was included. The effects of chlorophenol on aquatic organisms and humans were reviewed by Kozak *et al.*[15] Pentachlorophenol was by far the most toxic compound to fish with decreased growth occurring in sockeye salmon (*Oncorhynchus nerka*) at less than 2 μg/l. Available evidence indicated that moderate bioaccumulation and biomagnification of pentachlorophenol may occur in aquatic organisms.

Natural and artificial sources of radiation and their effects on aquatic life were reviewed by Ravera.[16] It was concluded that the level of environmental contamination by radioisotopes was too low to produce noticeable effects on aquatic biota including fish. Fromm[17] reviewed and discussed the current state of knowledge of the effect of pH (acid stress) to freshwater fish. The no-effect level appeared to be about pH 6.5 but acclimated species, such as trout, tolerated pH 5.0 for long periods. Sublethal, but debilitating effects appeared to be related to osmoregulation and oxygen-carrying ability of blood.

Impacts of navigational dredging on fish, other aquatic biota, and wildlife were reviewed by Allen and Hardy.[18] Discussions on impacts and habitat development were divided into coastal waters and rivers, with a limited discussion of the Great Lakes. Impacts on rivers and the Great Lakes included studies dealing with the effects of channelization and disposal of dredge material on freshwater fish and aquatic invertebrates. A review of research on effects of sediments on warmwater fish indicated that incubation of eggs was most affected by turbid conditions.[19] It was found that warmwater species adapted to turbid conditions had reproductive cycles that cor-

related with times of turbidity. Van der Schalie *et al.*[20] reviewed literature on the use of fish to biomonitor effluents. The authors described a system utilizing bluegill sunfish (*Lepomis macrochirus*) ventilation rates as indicators of effluent changes. The fish responded to a simulated acetone spill which illustrated the utility of the method.

WATER QUALITY

Dissolved gases. A series of bioassays designed to determine ozone levels and doses to prevent unwanted introduction of fishes into a reservoir was conducted by Coler and Asbury.[21] Larvae and, in some species, eggs were exposed in static and continuous tests to determine 24-d LC_{50} values (Table 1). Generally, the eggs of fish were found to be more tolerant than larvae to ozone concentrations. In another study,[22] eggs of yellow perch (*Perca flavescens*), white sucker (*Catostomus commersoni*), fathead minnow (*Pimephales promelas*), and bluegills (*Lepomis macrochirus*) were more tolerant of dissolved ozone than were the larvae.

Hazel *et al.*[23] published a monograph on experimental methods, data generation, data manipulation and rationale for development of water criteria for ammonia and total residual chlorine (TRC). The orange throated darter (*Etheostoma spectabile*), red shiner (*Notropis lutrensis*), the elmid beetle, and a stream crayfish were compared. The shiner was most sensitive to ammonia and the darter was most sensitive to chlorine. Safe levels were reported as 0.07 mg NH_3/l and as 0.02 mg TRC/l. Alabaster *et al.*[24] determined that an acclimation of 1 day at 0.15 mg NH_3/l increased the 24-h LC_{50} between 38 and 79% in Atlantic salmon (*Salmo salar*) smolts. Channel catfish (*Ictalurus punctatus*) exposed to ammonia in sodium chloride isonatremic test water displayed greater short-term resistance than in fresh water.[25] The 6-, 12-, and 24-h LC_{50} values were higher in the isonatremic water, but 48-h LC_{50} values were not different. In another study[26] the 96-h LC_{50} value of un-ionized ammonia ranged from 1.0 to 1.3 mg/l for the spotfin shiner (*Notropis spilopterus*). The authors noted that LC_{50} values for warmwater species were higher than those for coldwater species. Rankin[27] exposed sockeye salmon (*Oncorhynchus nerka*) eggs from fertilization to hatching to ammonia concentrations from 0.099 to 4.05 mg NH_3-N/l and determined a 62-d LC_{50} of 0.11 mg/l. At 0.099 mg NH_3-N/l, 9.4% hatch occurred before any control hatch. Both ammonia and pH (as elevated by Tris buffer to 8.4) resulted in smaller alevins than those in the control. The effect of ammonium chloride on predator-prey systems using brook trout (*Salvelinus fontinalis*) as a predator and juvenile chinook salmon (*Oncorhynchus tshawytscha*) as the prey was determined in an artificial stream.[28] Ammonium concentrations were varied from 1 to 0.76 mg/l while prey were maintained at three densities. Predation was depressed at 0.41 mg/l at all prey densities while predation increased with prey density. Five species of salmonid eggs and fry were exposed to four nitrate concentrations from 0 to 20 mg/l.[29] Five to 10 mg/l was mildly toxic to developing eggs and early fry of rainbow and steelhead trout (*Salmo gairdneri*). Chinook salmon and cutthroat trout (*Salmo clarki*) were affected at levels of 20 mg/l or more. Coho salmon (*Oncorhynchus kisutch*) were not affected at the exposures used.

Reynolds and Haines[30] exposed sac fry brown trout (*Salmo trutta*) to 0 to 13 μg/l of H_2S and obtained a 96-h LC_{50} value of 7 μg/l. Gill lamellae of fish from a 22-d exposure had lower length to diameter ratios than those from the controls. The inhibition of spermatogenesis by HCN was examined in rainbow trout.[31] Concentrations of 0.01 and 0.03 mg HCN/l reduced the number of dividing spermatogonia by 13 and 50%, respectively. Cellular damage was evident in spermatogonia in all phases of the cell cycle accompanied by a high incidence of necrosis at the higher HCN concentration.

The effect of DO concentration on the sensitivity of carp (*Cyprinus carpio*), guppy (*Poecilia reticulata*), and zebrafish (*Brachydanio rerio*) to detergents was investigated by Rieger.[32] Results showed that zebrafish were most sensitive, followed by guppies and carp. The effect of reduced DO was slight at high detergent concentrations, but large at low detergent concentrations. Carlson *et al.*[33] determined that channel catfish consumed less food and grew less at DO concentrations of 3.5 mg/l. Yellow perch consumed less food and growth was unaffected at 3.5 mg DO/l but both parameters were affected at 2.0 mg/l. Survival of both species was unaffected by reduced DO exposures. Lake herring (*Coregonus artedii*) were hatched in four temperatures (2 to 8°C) and five DO concentrations (1 to 12 mg/l).[34] Lower DO levels increased the time to median hatch, reduced mean total length of the fry, and decreased the percentage of normal fry. Evidence that embryos have a decreasing DO demand at lower temperatures was developed.

pH. Yolk-sac fry of brown trout exposed to snowmelt water from seven southern Norway localities were studied by Grande and Andersen.[35] The toxicity of the waters depended mainly on the pH (pH of the meltwater varied from 4.43 to 5.20), but low concentrations of dissolved salts were influential. Tests using eggs of Atlantic salmon exposed to acidified water indicated that hatching time was delayed at pH of 5.5 and below and no hatching occurred at pH of 4.0.[36] Egg mortality was correlated with pH. Eggs exposed to pH 4.0 to 4.2 for 5 to 7 days hatched when returned to normal water but with higher mortalities. Inhibition of the hatching enzyme, chorionase, was suggested. In other studies[37] embryos and alevins of three strains of Atlantic salmon and one strain of rainbow trout were tested for low pH tolerance. Acclimation to low pH did not appear to occur. All strains of alevins had 10 000-min LC_{50} values of pH 4.3 and were less tolerant than embryos. Studies of Atlantic salmon exposed to pH 6.8 (control), 9.0 and 9.5 from fertilization to 50 days after hatching showed that developmental processes and hatching were unaffected.[38] A pH of 9.5, however, caused a greater cumulative mortality (18%) than the controls (1.3%). Similar tests[39] with Atlantic salmon exposed to pH 3.7 to 6.8 from fertilization through 40 days after hatching indicated that lethal and sublethal injuries were separable only to the degree, distribution, and to the influence on different morphological features. Acidaemia in rainbow trout exposed to pH 4.0 water with and without CO_2 was determined not to be because of hypercapnia or of lactic acid accumulation[40]. However, electrolyte loss was attributed to ambient low pH.

CHEMICAL POLLUTANTS

Insecticides. Hubert[41] measured chlorinated hydrocarbon insecticide residues in carp (*Cyprinus carpio*) from impounded and riverine segments of the Des Moines River. Concentrations of dieldrin, DDE, DDD, and DDT in muscle tissue were mea-

Table 1—A summary of the acute and chronic toxicity of inorganic and organic pollutants to freshwater fish.

Pollutant[Ref]	Species	Results		Exposure type	Temp. (°C)	Test conditions	
Acephate[80]	Rainbow trout (*Salmo gairdneri*)	24-h LC_{50}	3 160 mg/l	S[a]	8	pH	8.0
		24-h LC_{50}	2 890 mg/l	S	15	HD[b]	90
		25-h LC_{50}	1 880 mg/l	S	22		
Acridine[300]	Fathead minnow (*Pimephales promelas*)	24-h BCF[c]	125	F[d]	22	pH	7.3–7.7
Acrolein[113]	Coho salmon (*Oncorhynchus kisutch*)	96-h LC_{50}	68 μg/l	S	10		—
Actusol (T-766)[208]	Rainbow trout	96-h LC_{50}	14 mg/l	S	15	pH	6.1–7.1
						HD	12.1–24.4
Alkyl ethoxysulfate (C12-15)[266]	Brown trout (*Salmo trutta*)	96-h LC_{50}	1.5 mg/l	R[e]	15	HD	250
		96-h LC_{50}	1.0–2.5 mg/l	R	15	HD	26–30
	Golden orfe (*Idus idus melanotus*)	96-h LC_{50}	6.2 mg/l	R	20	HD	268
		96-h LC_{50}	3.3 mg/l	S	20		
Alkyl olefin sulfonate (chain length C14-16)[266]	Harlequin fish (*Rasbora heteromorpha*)	96-h LC_{50}	3.3 mg/l	R	20	HD	20
	Brown trout	96-h LC_{50}	3.1 mg/l	R	15	HD	250
		96-h LC_{50}	2.5–5.0 mg/l	R	15	HD	26–30
	Golden orfe	96-h LC_{50}	4.9 mg/l	R	20	HD	268
		96-h LC_{50}	3.4 mg/l	S	20		
Alkyl olefin sulfonate (chain length C16-18)[266]	Harlequin fish	96-h LC_{50}	0.5 mg/l	R	20	HD	20
	Brown trout	96-h LC_{50}	0.5 mg/l	R	15	HD	250
	Golden orfe	96-h LC_{50}	0.9 mg/l	R	20	HD	268
Aminocarb[54]	Atlantic salmon (*Salmo salar*) juvenile	96-h LC_{50}	8.7 mg/l	S	10		—
Amitrole-T[113]	Coho salmon	96-h LC_{50}	70 mg/l	S	10	pH	7.5–7.6
						HD	100
Ammonia[24]	Atlantic salmon-smolts	24-h LC_{50}	0.14–0.50 mg/l	—	12	pH	7.7
						HD	264
Ammonia[25]	Channel catfish (*Ictalurus punctatus*)	24-h LC_{50} (Freshwater)	270 mg/l	—	20–24	pH	6.9–7.1
		24-h LC_{50} (NaCl added)	338 mg/l	—	20–24	HD	47
		48-h LC_{50} (Freshwater)	288 mg/l	—	20–24		
		48-h LC_{50} (NaCl added)	257 mg/l	—	20–24		
Ammonia (un-ionized)[27]	Sockeye salmon (*Oncorhynchus nerka*)-eggs	62-d LC_{50}	0.11 mg/l	—	10	pH	8.4
Ammonia (un-ionized)[26]	Spotfin shiner (*Notropis spilopterus*)	96-h LC_{50}	1.0–1.3 mg/l	F	27	pH	7.7–8.5
						HD	82

Ammonia (un-ionized)[212]	Channel catfish	24-h LC_{50}	1.39–1.82 mg/l	S	21–25	pH	7.0–9.0
						HD	40
Ammonia (un-ionized)[356]	Rainbow trout	96-h LC_{50}	0.77 mg/l	F	14	pH	8.0
	Fathead minnow	96-h LC_{50}	1.59 mg/l	F	14	HD	655
Analine[268]	Channel catfish						
	embryos-larvae	4-d LC_{50}	5.0–5.5 mg/l	F	26–30	pH	7.7
						HD	47
	embryos-larvae	4-d LC_{50}	6.2–6.3 mg/l	F	26–30	pH	7.7
						HD	195
Aniline[268]	Largemouth bass (*Micropterus salmoides*)-embryos-larvae	4-d LC_{50}	12–33 mg/l	F	18–26	pH	7.7
						HD	47
	Goldfish (*Carassius auratus*)-embryos-larvae	4-d LC_{50}	5.5–9.3 mg/l	F	18–26		
	Largemouth bass						
	embryos-larvae	4-d LC_{50}	7–30 mg/l	F	18–26	pH	7.7
						HD	195
	Goldfish						
	embryos-larvae	4-d LC_{50}	4.6–7.6 mg/l	F	18–26		
Anti-Germ 50 (cationic polyelectrolyte)[112]	*Tilapia nilotica*	96-h LC_{50}	3.8 mg/l	S	24	pH	7.5–7.7
						HD	120–133
Antimycin A[128]	American eel (*Anguilla rostrata*)						
	black eel stage	96-h LC_{50}	0.28 µg/l	S	22	pH	7.2–7.6
						HD	40–48
Antimycin A[129]	American eel						
	glass eel stage	96-h LC_{50}	0.09 µg/l	S	22	pH	7.2–7.6
						HD	40–48
Antimycin A[130]	American eel						
	yellow phase	96-h LC_{50}	3 µg/l	S	22	pH	7.2–7.6
						HD	40–48
Arsenic (arsenic trioxide)[193]	Ciscoe (*Coregonus sp.*) (hybrid)	96-h LC_{50}	17 mg/l	S	7–8	HD	40–48
Arsenic (arsenic trioxide)[168]	*Puntius sophore*	48-h LC_{50}	18 mg/l	S	35	pH	7.2
Asulam[109]	Grass carp (*Ctenopharyngodon idella*)	96-h LC_{50}	>10 000 mg/l	S	18–21	pH	8.1
						HD	270
Atrazine[268]	Rainbow trout						
	embryos	23-d LC_{50}	0.92 mg/l	F	13–15	pH	8.0
	larvae	4-d LC_{50}	0.87 mg/l	F	13–15	HD	51
	Channel catfish						
	embryos-larvae	4-d LC_{50}	0.22–0.34 mg/l	F	26–30		
	Rainbow trout						
	embryos	23-d LC_{50}	1.11 mg/l	F	13–15	pH	7.9
	larvae	4-d LC_{50}	1.08 mg/l	F	13–15	HD	202
	Channel catfish						
	embryos-larvae	4-d LC_{50}	0.24–0.31 mg/l	F	26–30		

Table 1—(Continued)

Pollutant[Ref.]	Species	Results		Exposure type	Temp. (°C)	Test conditions	
Atrazine[113]	Coho salmon	96-h LC_{50}	>15 mg/l	S	10	pH HD	7.4–7.6 101
Atrazine[102]	Bluegill (*Lepomis macrochirus*)	96-h LC_{50}	15 mg/l	S	—		—
p-Benzoquinone[292]	Rainbow trout	96-h LC_{50}	0.125 mg/l	F	14	pH	8.0
	Fathead minnow	96-h LC_{50}	0.045 mg/l	F	14	HD	707
BHC (technical grade)[50]	Mosquitofish (*Gambusia affinis*)	96-h LC_{50}	1 350 µg/l	S	27	pH HD	7.5 32
BP1100X[288]	Rainbow trout	96-h LC_{50}	3 600 mg/l	S	15	pH HD	6.1–7.1 12–24
Bromine (sodium bromide)[174]	Guppy (*Poecilia reticulata*)	125-d EC_{50}	180–225 mg/l	R	25		—
Cadmium[171]	Rainbow trout	48-h LC_{50}	0.09 mg/l	S	16	pH HD	7.2 20
		48-h LC_{50}	0.36 mg/l	S	16	pH HD	7.2 80
		48-h LC_{50}	3.70 mg/l	S	16	pH HD	7.2 320
Cadmium[214]	Goldfish	96-h LC_{50}	12.6 mg/l	F	25	HD	99
Cadmium[181]	Northern squawfish (*Ptychocheilus oregonensis*) juvenile	96-h LC_{50}	1 092–1 104 µg/l	F	12–17	pH	7.1–7.5
Cadmium[199]	Atlantic salmon	4.5-mo. MATC[f]	1–5 µg/l	—	—	pH HD	6.3 13
Cadmium chloride[174]	*Oryzias latipes*	13-d EC_{50}	20 µg/l	R	24		—
	Xenopus laevis	100-d EC_{50}	650 µg/l	R	20		—
Cadmium sulfate[217]	*Colisa fasciatus*	96-h LC_{50}	126 mg/l	S	25	pH HD	7.3 120
Capacitor 21 (PCB)[258]	Redear sunfish (*Lepomis microlophus*) embryos-larvae	4-d LC_{50}	0.013–0.019 mg/l	F	18–26	pH HD	7.8 50
	Largemouth bass embryos-larvae	4-d LC_{50}	0.001 5–0.0023 mg/l	F	18–26		
	Redear sunfish embryos-larvae	4-d LC_{50}	0.008–0.014 mg/l	F	18–26	pH HD	7.6 204
	Largemouth bass embryos-larvae	4-d LC_{50}	0.001 8–0.0027 mg/l	F	18–26		
	Rainbow trout embryos	23-d LC_{50}	0.002 0 mg/l	F	13–15		
	larvae	4-d LC_{50}	0.002 0 mg/l	F	13–15		

Carbofuran[102]	Bluegill	96-h LC_{50}	0.25 mg/l	S	—		—
Carbofuran (75% W.P.)[56]	*Mystus vittatus*	96-h LC_{50}	0.31 mg/l	—	—		—
Catechol[292]	Rainbow trout	96-h LC_{50}	8.9 mg/l	F	140	pH	8.0
	Fathead minnow	96-h LC_{50}	3.5 mg/l	F	140	HD	707
Cationic detergent[266]	Harlequin fish	96-h LC_{50}	0.5 mg/l	R	20	HD	20
	Brown trout	96-h LC_{50}	1.0 mg/l	R	15	HD	26–30
	Golden orfe	96-h LC_{50}	1.6 mg/l	S	20	HD	268
Chloramines[349]	Goldfish	96-h LC_{50}	0.35 mg/l	F	21	pH	7.3
						HD	52
Chlorfenvinphos[96]	Guppy	96-h LC_{50}	0.6–0.9 mg/l	S	22		—
Chlorine[348]	Rainbow trout	Lethal	0.03–0.09 mg/l	F	10–25		—
Chlorine[340]	Fathead minnow juvenile	96-h LC_{50}	0.120 mg/l	F	25	pH	7.2–7.6
						HD	700
	Pugnose shiner (*Notropis anogenus*) juvenile	96-h LC_{50}	0.029 mg/l	F	26		
	Pugnose minnow (*Opsopoeodus emiliae*) juvenile	96-h LC_{50}	0.085 mg/l	F	25		
	Goldfish juvenile	96-h LC_{50}	0.519 mg/l	F	13		
	Brook trout (*Salvelinus fontinalis*) juvenile	96-h LC_{50}	0.068 mg/l	F	16		
	Rainbow trout juvenile	96-h LC_{50}	0.037 mg/l	F	16		
	Splake (*Salvenlinus* sp.) juvenile	96-h LC_{50}	0.071 mg/l	F	15		
	Chinook salmon (*Oncorhynchus tshawytscha*) juvenile	96-h LC_{50}	0.065 mg/l	F	15		
o-Chlorobenzaldehyde[296]	Rainbow trout juvenile	96-h LC_{50}	2.45 mg/l	F	15	pH	7.1–7.4
						HD	290
Chlorobenzene[268]	Goldfish embryos-larvae	4-d LC_{50}	0.88–3.48 mg/l	F	18–26	pH	7.6
						HD	51
	Largemouth bass embryos-larvae	4-d LC_{50}	0.05–0.34 mg/l	F	18–26		
	Rainbow trout embryos	23-d LC_{50}	<0.09 mg/l	F	13–15		
	Goldfish embryos-larvae	4-d LC_{50}	1.04–2.37 mg/l	F	18–26	pH	7.6
						HD	203
	Largemouth bass embryos-larvae	4-d LC_{50}	0.06–0.39 mg/l	F	18–26		

Table 1—(Continued)

Pollutant[Ref.]	Species	Results	Exposure type	Temp. (°C)	Test conditions	
	Rainbow trout embryos	23-d LC_{50}	<0.09 mg/l	F	13–15	
o-Chlorobenzylidenemalonotrile (CS gas)[296]	Rainbow trout juvenile	96-h LC_{50}	0.22 mg/l	F	15	pH 7.1–7.4
Chloroform[268]	Rainbow trout					HD 209
	embryos	23-d LC_{50}	2.03 mg/l	F	13–15	pH 7.3
	larvae	4-d LC_{50}	2.03 mg/l	F	13–15	HD 49
	embryos	23-d LC_{50}	1.24 mg/l	F	13–15	pH 7.3
	larvae	4-d LC_{50}	1.24 mg/l	F	13–15	HD 210
4-Chloro-o-cresol[294]	Brown trout	24-h LC_{50}	2.12 mg/l	R	10	—
9-Chlorophenanthrene[361]	Fathead minnow	28-d BCF	5 000	F	24	—
4-Chlororesorcinol[281]	Carp (*Cyprinus carpio*) embryo	4- to 7-d LC_{50}	10–100 mg/l	R	24	—
Chromium (VI)[175]	Fathead minnow					
	juvenile	96-h LC_{50}	36.2 mg/l	S	25	pH 7.7
	juvenile	96-h LC_{50}	36.9 mg/l	F	25	HD 209
	life cycle	MATC	1.0–4.0 mg/l	F	16–24	
Chromium trioxide[224]	*Colisa fasciatus*	96-h LC_{50}	40 mg/l	S	25	pH 7.3
Copper[203]	Pumpkinseed (*Lepomis gibbosus*)					HD 120
	junvenile	96-h LC_{50}	1.24–1.67 mg/l	F	20	pH 7.2
	adult	96-h LC_{50}	1.74–1.94 mg/l	F	20	HD 125
	Rainbow trout					
	juvenile	96-h LC_{50}	0.19–0.20 mg/l	F	10	pH 7.8
Copper[177]	adult	96-h LC_{50}	0.21 mg/l	F	10	HD 125
	Bluntnose minnow					
	(*Pimephales notatus*)	96-h LC_{50}	230 µg/l	F	25	pH 7.9–8.3
Copper[181]		9-mo MATC	4.3–18.0 µg/l	F	25	HD 200
	Northern squawfish					
	juvenile	96-h LC_{50}	23 µg/l	F	8	pH 7.1–7.5
	Northern squawfish					HD 20–30
	Chiselmouth (*Acrocheilus alutaceus*)	96-h LC_{50}	18 µg/l	F	12	pH 7.1–7.5
Copper[183]	Bluegill	96-h LC_{50}	143 µg/l	F	11	HD 52–56
		96-h LC_{50}	0.9–1.1 mg/l	F	19–22	pH 6.8–7.5
Copper nitrate[225]	*Colisa fasciatus*					HD 21–59
	adult females	96-h LC_{50}	4.0 mg/l	S	25	pH 7.3
						HD 120

Copper nitrate[50]	Mosquitofish	96-h LC_{50}	93 µg/l	S	27	pH	7.5
						HD	32
Copper oxychloride[50]	Mosquitofish	96-h LC_{50}	1 750 µg/l	S	27	pH	7.5
						HD	32
Copper sulfate[349]	Goldfish	96-h LC_{50}	0.30 mg/l	F	21	pH	7.3
						HD	52
Copper sulfate[185]	*Poecilia reticulata*	48-h LC_{50}	36.4 mg/dm^3	S	—		—
Copper sulfate (hydrated)[180]	*Poecilia reticulata*	96-h LC_{50}	160–480 µg/l	R	17–22	pH	6.4–6.6
	Labeo rohita	96-h LC_{50}	87–295 µg/l	R	17–22	HD	144–188
	Carp	96-h LC_{50}	117–530 µg/l	R	17–22		
Copper sulfate[182]	*Puntius conchonius*	96-h LC_{50}	0.57 mg/l	S	13–22	pH	8.1
						HD	310
Copper sulfate[128]	American eel black eel stage	96-h LC_{50}	3.20 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Copper sulfate[129]	American eel glass eel stage	96-h LC_{50}	2.54 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Copper sulfate[50]	Mosquitofish	96-h LC_{50}	200 µg/l	S	27	pH	7.5
						HD	32
Copper + Zinc[184]	Chinook salmon fry	96-h LC_{50}	20–37 µg Cu/l + 132–279 µg Zn/l	F	9–11	pH	6.6–7.4
						HD	21–27
Corexit 19-L-50[288]	Rainbow trout	96-h LC_{50}	380 mg/l	S	15	pH	6.1–7.1
						HD	12–24
Corexit 8666[288]	Rainbow trout	96-h LC_{50}	>10 000 mg/l	S	15	pH	6.1–7.1
						HD	12–24
Corexit 9517[288]	Rainbow trout	96-h LC_{50}	180 mg/l	S	15	pH	6.1–7.1
						HD	12–24
Corexit 9527[288]	Rainbow trout	96-h LC_{50}	260 mg/l	S	15	pH	6.1–7.1
						HD	12–24
m-Cresol[292]	Rainbow trout	96-h LC_{50}	8.9 mg/l	F	14	pH	8.0
	Fathead minnow	96-h LC_{50}	55.9 mg/l	F	14	HD	707
o-Cresol[292]	Rainbow trout	96-h LC_{50}	8.4 mg/l	F	14	pH	8.0
	Fathead minnow	96-h LC_{50}	18.2 mg/l	F	14	HD	707
p-Cresol[292]	Rainbow trout	96-h LC_{50}	7.9 mg/l	F	14	pH	8.0
	Fathead minnow	96-h LC_{50}	28.6 mg/l	F	14	HD	707
Cutrin[107]	Fathead minnow	96-h LC_{50}	0.21 mg/l	S	18	pH	7.7
		96-h LC_{50}	0.12 mg/l	S	14		
	Largemouth bass	96-h LC_{50}	6.4 mg/l	S	21	pH	7.3

Table 1—(Continued)

Pollutant[Ref.]	Species		Results	Exposure type	Temp. (°C)	Test conditions	
Cyanatryn (Aqualin)[109]	Grass carp	96-h LC_{50}	58 mg/l	F	18–21	pH	8.1
						HD	270
2,4-D[268]	Goldfish						
	embryos-larvae	4-d LC_{50}	133–>187 mg/l	F	18–26	pH	7.8
						HD	51
	Largemouth bass						
	embryos-larvae	4-d LC_{50}	109–165 mg/l	F	18–26		
	Rainbow trout						
	embryos	23-d LC_{50}	11 mg/l	F	13–15		
	larvae	4-d LC_{50}	11 mg/l	F	13–15		
	Goldfish						
	embryos-larvae	4-d LC_{50}	119–>201 mg/l	F	18–26	pH	7.7
						HD	200
	Largemouth bass						
	embryos-larvae	4-d LC_{50}	82–161 mg/l	F	18–26		
	Rainbow trout						
	embryos	23-d LC_{50}	4.2 mg/l	F	13–15		
	larvae	4-d LC_{50}	4.2 mg/l	F	13–15		
2,4-D[113]	Coho salmon	96-h LC_{50}	>200 mg/l	S	10	pH	7.2–7.6
						HD	105
2,4-D (amine)[109]	Grass carp	96-h LC_{50}	1 313 mg/l	F	18–21	pH	8.1
						HD	270
Dalapon[109]	Grass Carp	96-h LC_{50}	>30 000 mg/l	S	18–21	pH	8.1
						HD	270
DDT[50]	Mosquitofish	96-h LC_{50}	40 µg/l	S	27	pH	7.5
						HD	32
Dechlorane 602[60]	Atlantic salmon						
	juvenile	96-h BCF	7.2	S	—		—
Dicamba[113]	Coho salmon	96-h LC_{50}	>100 mg/l	S	10	pH	7.7–7.8
						HD	102
Dichlobenil[109]	Grass carp	96-h LC_{50}	9.4 mg/l	S	18–21	pH	8.1
						HD	270
1,4-Dichlorobenzene[304]	Guppy	2-d BCF	1 800	F	21		—
3,3'-Dichlorobenzidine[306]	Bluegill	96-h BCF	495	F	21		—
		168-h BCF	507	F	21		—
2,4-Dichlorophenol[268]	Goldfish						
	embryos-larvae	4-d LC_{50}	0.39–1.76 mg/l	F	18–26	pH	7.8
						HD	50
	Channel catfish						
	embryos-larvae	4-d LC_{50}	1.35–1.85 mg/l	F	26–30		

	Rainbow trout						
	embryos	23-d LC_{50}	0.08 mg/l	F	13–15		
	larvae	4-d LC_{50}	0.08 mg/l	F	13–15		
	Goldfish						
	embryos-larvae	4-d LC_{50}	0.26–1.24 mg/l	F	18–26	pH	7.8
						HD	200
	Channel catfish						
	embryos-larvae	4-d LC_{50}	1.07–1.70 mg/l	F	26–30		
	Rainbow trout						
	embryos	23-d LC_{50}	0.07 mg/l	F	13–15		
	larvae	4-d LC_{50}	0.07 mg/l	F	13–15		
Dichlorvos[51]	*Tilapia mossambica*	96-h LC_{50}	1.4–1.9 mg/l	S	29	pH	7.0–7.5
Dichlorvos (76% EC)[56]	*Mystus vittatus*	96-h LC_{50}	0.45 mg/l	—	—		—
Dinoseb (Premerge)[113]	Coho salmon	24-h LC_{50}	190 µg/l	S	10	pH	7.7–7.8
		144-h LC_{50}	88 µg/l	S	10	HD	101
Dioctyl phthalate[268]	Goldfish						
	embryos-larvae	4-d LC_{50}	>186 mg/l	F	18–26	pH	7.5
						HD	53
	Largemouth bass						
	embryos-larvae	4-d LC_{50}	55.7–65.5 mg/l	F	18–26		
	Rainbow trout						
	embryos	23-d LC_{50}	139.1 mg/l	F	13–15		
	larvae	4-d LC_{50}	139.5 mg/l	F	13–15		
	Goldfish						
	embryos-larvae	4-d LC_{50}	>191 mg/l	F	18–26	pH	7.4
						HD	205
	Largemouth bass						
	embryos-larvae	4-d LC_{50}	32.1–45.4 mg/l	F	18–26		
	Rainbow trout						
	embryos	23-d LC_{50}	154 mg/l	F	13–15		
	larvae	4-d LC_{50}	149.2 mg/l	F	13–15		
Diquat[109]	Grass carp	96-h LC_{50}	1.718 mg/l	F	18–21	pH	8.1
						HD	270
Diquat[113]	Coho salmon	96-h LC_{50}	30 mg/l	S	10		—
		144-h LC_{50}	19 mg/l	S	10		—
Diquat[138]	American eel						
	black eel stage	96-h LC_{50}	55.4 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Diuron[109]	Grass carp	96-h LC_{50}	31 mg/l	F	13	pH	8.1
						HD	270
Drew Chemical OSE 71[268]	Rainbow trout	96-h LC_{50}	3 200 mg/l	S	15	pH	6.1–7.1
						HD	12–24
Drew Chemical OSE 72[268]	Rainbow trout	96-h LC_{50}	3 200 mg/l	S	15	pH	6.1–7.1
						HD	12–24

Table 1—(Continued)

Pollutant[Ref]	Species	Results		Exposure type	Temp. (°C)	Test conditions	
Duosol[269]	Rainbow trout	96-h LC_{50}	<24 mg/l	S	15	pH HD	6.1-7.1 12-24
Dylox[129]	American eel black eel stage	96-h LC_{50}	1.32 mg/l	S	22	pH HD	7.2-7.6 40-48
Dylox[129]	American eel glass eel stage	96-h LC_{50}	1.31 mg/l	S	22	pH HD	7.2-7.6 40-48
Dylox[130]	American eel yellow phase	96-h LC_{50}	8.57 mg/l	S	22	pH HD	7.2-7.6 40-48
Endosulfan (4% dust)[59]	*Labeo rohita*	96-h LC_{50}	1.25 µg/l	F	28	pH HD	8.4 152
Endosulfan (Thiodan 35 percent EC)[50]	Mosquitofish	96-h LC_{50}	3.2 µg/l	S	27	pH HD	7.5 32
Endosulfan (35% EC)[58]	*Mystus vittatus*	96-h LC_{50}	0.67 µg/l	—	—		—
Endosulfan (35% EC)[59]	*Labeo rohita*	96-h LC_{50}	1.0 µg/l	F	28	pH HD	8.4 152
Endosulfan (Isomer A)[60]	*Labeo rohita*	96-h EC_{50}	0.33 µg/l	F	28	pH HD	8.4 152
Endosulfan (Isomer B)[59]	*Labeo rohita*	96-h LC_{50}	7.1 µg/l	F	28	pH HD	8.4 152
Endosulfan (96% technical grade)[60]	*Labeo rohita*	96-h LC_{50}	1.1 µg/l	F	28	pH HD	8.4 152
Endosulfan (technical grade)[50]	Mosquitofish	96-h LC_{50}	8.0 µg/l	S	27	pH HD	7.5 32
Endrin[76]	*Ophiocephalus punctatus*	96-h LC_{50}	33 µg/l	S	29	pH HD	6.9 150
Endrin[82]	Catfish (*Heteropneustes fossilis*) gravid female	96-h LC_{50}	20 µg/l	S	24-27		—
Endrin (91.1%)[50]	Black bullhead (*Ictalurus melas*)	96-h LC_{50} 28-d LC_{50} 4-d BCF 7-d BCF	0.45 µg/l 0.10 µg/l 3 700 6 200	F F F F	15 15 15 15	pH HD	7.6-7.8 46-48
Energy Plus[269]	Rainbow trout	96-h LC_{50}	57 mg/l	S	15	pH HD	6.1-7.1 12-24

Esteron[113]	Coho salmon	96-h LC_{50}	>800 mg/l	S	10	pH	7.4
						HD	100
Fenitrothion[80]	Rainbow trout	24-h LC_{50}	4.3 mg/l	S	8	pH	8.0
		24-h LC_{50}	2.7 mg/l	S	15	HD	90
		24-h LC_{50}	1.9 mg/l	S	22		
Formalin[126]	American eel black eel stage	96-h LC_{50}	224 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Formalin[125]	American eel glass eel stage	96-h LC_{50}	84 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Formalin (37% formaldehyde)[130]	American eel yellow phase	96-h LC_{50}	330 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Fosamine (ammonium salt)[114]	Channel catfish	28-d BCF	<1.0	S	21	pH	6.0
Furanace[128]	American eel black eel stage	96-h LC_{50}	0.96 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Furanace[129]	American eel glass eel stage	96-h LC_{50}	0.77 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Furanace[131]	Channel catfish	96-h LC_{50}	0.94 mg/l	S	21	pH	7.6
						HD	50
Glyphosate[109]	Grass carp	96-h LC_{50}	15 mg/l	F	18–21	pH	8.1
						HD	270
Hexachlorobenzene[304]	Guppy female	7-d BCF	290 000	F	21		—
Hydrazine[282]	Bluegill	96-h LC_{50}	1.08 mg/l	S	23–24	pH	7.2–8.4
						HD	240–292
Hydrogen sulphide[30]	Brown trout (*Salmo trutta*)	96-h LC_{50}	7.0 µg/l	F	13	pH	7.1
						HD	127
Hydroquinone[282]	Rainbow trout	96-h LC_{50}	0.097 mg/l	F	14	pH	8.0
	Fathead minnow	96-h LC_{50}	0.044 mg/l	F	14	HD	707
Imbetin-N/52 (commercial non-ionic surfactant)[290]	Rainbow trout 1-yr-old	24-h LC_{50}	8.5 mg/l	S	15	pH	7.5–8.1
						HD	114
Iron ($FeCl_3$)[185]	*Poecilia reticulata*	48-h LC_{50}	73 mg/dm³	S	—		—
Landfill leachate[305]	Rainbow trout	96-h LC_{50}	5.8–7.5‰	S	15		—
LAS[266]	Harlequin fish	96-h LC_{50}	4.6 mg/l	R	20	HD	20
	Brown trout	96-h LC_{50}	1.4 mg/l	R	15	HD	26–30

Table 1—(Continued)

Pollutant[Ref]	Species	Results		Exposure type	Temp. (°C)	Test conditions	
	Golden orfe	96-h LC_{50}	1.2 mg/l	R	20	HD	268
		96-h LC_{50}	1.3 mg/l	S	20		
LAS (15.4 percent active material)[288]	Harlequin fish	96-h LC_{50}	6.1 mg/l	R	20	HD	20
	Brown trout	96-h LC_{50}	0.9 mg/l	R	15	HD	250
	Golden orfe	96-h LC_{50}	4.6 mg/l	R	15	HD	26–30
		96-h LC_{50}	1.9 mg/l	R	20	HD	268
		96-h LC_{50}	2.9 mg/l	S	20		
LAS (chain length C10-15)[288]	Harlequin fish	96-h LC_{50}	0.7 mg/l	R	20	HD	20
	Brown trout	96-h LC_{50}	0.1 mg/l	R	15	HD	250
	Golden orfe	96-h LC_{50}	0.25–0.50 mg/l	R	15	HD	26–30
		96-h LC_{50}	0.4 mg/l	R	20	HD	268
		96-h LC_{50}	0.6 mg/l	S	20		
LAS (chain length 11.2)[288]	Fathead minnow	96-h LC_{50}	12.3 mg/l	S	22	pH	7.2
		270-d MATC	5.1–8.4 mg/l	F	22	HD	39
LAS (chain length 11.7)[299]	Fathead minnow	96-h LC_{50}	4.1 mg/l	S	22	pH	7.2
		270-d MATC	0.48 mg/l	F	22	HD	39
LAS (chain length 13.3)[299]	Fathead minnow	96-h LC_{50}	0.86 mg/l	S	22	pH	7.2
		270-d MATC	0.11–0.25 mg/l	F	22	HD	39
Linco-6[288]	Rainbow trout	96-h LC_{50}	>1 000 mg/l	S	15	pH	6.1–7.1
						HD	12–24
Linear alkylate sulfonate[349]	Goldfish	96-h LC_{50}	6.17 mg/l	F	21	pH	7.3
						HD	52
Malachite green[128]	American eel black eel stage	96-h LC_{50}	0.54 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Malachite green[129]	American eel glass eel stage	96-h LC_{50}	0.27 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Malachite green[130]	American eel yellow phase	96-h LC_{50}	2.86 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Malathion[92]	Indian catfish	96-h LC_{50}	45 mg/l	S	24–27		—
		96-h LC_{50}	38–42 mg/l	R	24–27		—
Malathion[288]	Goldfish embryos-larvae	4-d LC_{50}	1.20–2.61 mg/l	F	18–26	pH	7.7
						HD	54
	embryos-larvae	4-d LC_{50}	1.65–3.15 mg/l	F	18–26	pH	7.6
						HD	197

Malathion (technical grade)[52]	*Tilapia mossambica*	48-h LC_{50}	5.6 mg/l	S	—		—
Malononitrile[266]	Rainbow trout juvenile	96-h LC_{50}	1.63 mg/l	F	15	pH	7.1–7.4
						HD	290
Matacil[54]	Atlantic salmon juvenile	96-h LC_{50}	3.5 mg/l	S	10		—
Mercuric chloride[50]	Mosquitofish	96-h LC_{50}	180 μg/l	S	27	pH	7.5
						HD	32
Mercuric chloride[160]	*Poecilia reticulata*	96-h LC_{50}	30–54 μg/l	R	17–22	pH	7.8–8.1
Mercuric chloride[190]	*Labeo rohita*	96-h LC_{50}	15–184 μg/l	R	17–22	HD	144–188
Mercuric chloride[239]	Japanese ricefish (Oryzias latipes) Embryos	8-h LC_{50}	5.9–6.5 mg/l	S	24	pH	7.3
Mercuric chloride[169]	*Channa punctatus*	96-h LC_{50}	1.8 mg/l	S	18	pH	7.3
Mercury[243]	Chingatta (*Channa gachua*)	96-h LC_{50}	1.4 mg/l	S	24–25	HD	160
	Heteropneustes fossiles	96-h LC_{50}	0.35 mg/l	S	28	pH	7.5
Methoxy ethyl mercuric chloride[50]	*Sarotherodon mossambica* Mosquitofish	96-h LC_{50}	0.075 mg/l	S	28		—
Mirex[60]		96-h LC_{50}	910 μg/l	S	27	pH	—
	Atlantic salmon juvenile					HD	7.5
Misco ("No Flash")[268]	Rainbow trout	96-h BCF	84	S	—		32
		96-h LC_{50}	18 mg/l	S	15	pH	—
Momar OSR[268]	Rainbow trout	96-h LC_{50}	<10 mg/l	S	15	HD	6.1–7.1
Naphthalene[266]	Coho salmon fry	96-h LC_{50}	3.22 mg/l	F	—	pH	12–24
1-Naphthol[55]	*Labeo rohita*	96-h LC_{50}	2.6–3.13 mg/l	F	28	HD	6.1–7.1
Nickel sulfate[247]	Coho salmon juvenile	14-d LC_{50}	11.2 mg/l	F	15		12–24
n-Nitrosodethylamine[280]	Fathead minnow	96-h LC_{50}	775 mg/l	S	22–23	pH	—
n-Nitrosodimethylamine[280]	Fathead minnow	96-h LC_{50}	940 mg/l	S	22	HD	7.6–8.2
Nokomis-3[268]	Rainbow trout	96-h LC_{50}	75 mg/l	S	15	pH	65–78
Nonylphenol[54]	Atlantic salmon juvenile	96-h LC_{50}	0.9 mg/l	S	10	HD	8.1–8.3
						pH	288
						HD	8.1–8.3
						pH	202
						HD	6.1–7.1
							12–24
							—

Table 1—(Continued)

Pollutant[Ref]	Species		Results	Exposure type	Temp. (°C)	Test conditions	
Nonylphenol ethoxylate[266]	Harlequin fish	96-h LC_{50}	8.6 mg/l	R	20	HD	20
	Brown trout	96-h LC_{50}	1.0 mg/l	R	15	HD	26–30
	Golden orfe	96-h LC_{50}	7.0 mg/l	R	20	HD	268
		96-h LC_{50}	11.2 mg/l	S	20		
Noxfish (5% Rotenone)[128]	American eel black eel stage	96-h LC_{50}	50.5 µg/l	S	22	pH	7.2–7.6
						HD	40–48
Noxfish (5% Rotenone)[129]	American eel glass eel stage	96-h LC_{50}	15.3 µg/l	S	22	pH	7.2–7.6
						HD	40–48
Noxfish (5% Rotenone)[123]	Golden shiner (*Notemigonus crysoleucas*)	24-h LC_{50}	0.05–0.41 mg/l	S	20	pH	7.5–7.7
NTA[288]	Channel catfish embryos-larvae	4-d LC_{50}	329–388 mg/l	F	26–30	pH	8.1
						HD	52
	Goldfish embryos-larvae	4-d LC_{50}	240–270 mg/l	F	18–26		
	Rainbow trout embryos	23-d LC_{50}	92.3 mg/l	F	13–15		
	larvae	4-d LC_{50}	90.5 mg/l	F	13–15		
	Channel catfish embryos-larvae	4-d LC_{50}	385–394 mg/l	F	26–30	pH	7.9
						HD	187
	Goldfish embryos-larvae	4-d LC_{50}	243–257 mg/l	F	18–26		
	Rainbow trout embryos	23-d LC_{50}	120.1 mg/l	F	13–15		
	larvae	4-d LC_{50}	114 mg/l	F	13–15		
Oilsperse 43[288]	Rainbow trout	96-h LC_{50}	>1 000 mg/l	S	15	pH	6.1–7.1
						HD	12–24
Ozone[21]	Yellow perch (*Perca flavescens*) larvae	24-h LC_{50}	0.21 mg/l	S	10	pH	6.9
	eggs	24-h LC_{50}	>2.06 mg/l	S	10	HD	34
	Rainbow trout larvae	24-h LC_{50}	0.19–0.31 mg/l	S	10		
	Bluegill larvae	24-h LC_{50}	0.33 mg/l	S	21		
	American shad (*Alosa sapidissima*) eggs	24-h LC_{50}	0.39 mg/l	S	22		
	Channel catfish larvae	24-h LC_{50}	0.47 mg/l	S	26		
	eggs	24-h LC_{50}	4.0 mg/l	S	26		

Ozone (cont.)[21]	Spottail shiner (*Notropis hudsonius*) post larvae	24-h LC_{50}	1.22 mg/l	S	25	pH	6.9
						HD	34
Ozone[22]	Yellow perch larvae	2- to 5-min LC_{50}	<0.1 mg/l	F	17		—
	eggs	80-min LC_{50}	1.57–1.98 mg/l	F	14		—
	White sucker (*Catostomus commersoni*) eggs	80-min LC_{50}	1.43 mg/l	F	14		—
	Fathead minnow larvae	0.5 to 5-min LC_{50}	<0.1 mg/l	F	24		—
	eggs	160-min LC_{50}	1.1 mg/l	F	24		—
	Bluegill larvae	0.5- to 2-min LC_{50}	<0.1 mg/l	F	26		—
Paraquat[109]	Grass carp	96-h LC_{50}	66 mg/l	F	18–21	pH	8.1
						HD	270
Paraquat[113]	Coho salmon	96-h LC_{50}	76 mg/l	S	10		—
PCB (Aroclor 1254)[193]	Ciscoe (hybrid)	96-h LC_{50}	>10 mg/l	S	7–8	HD	40–48
Pentachlorobenzene[304]	Guppy female	7-d BCF	260 000	F	21		—
Pentachlorophenol[189]	Chingatta	96-h LC_{50}	0.39 mg/l	S	24–25	pH	7.5
Phenanthrene[361]	Fathead minnow juvenile	28-d BCF	3 100–5 100	F	24		—
Phenol[292]	Rainbow trout	96-h LC_{50}	8.9 mg/l	F	14	pH	8.0
	Fathead minnow	96-h LC_{50}	67.5 mg/l	F	14	HD	707
		96-h LC_{50}	24.9 mg/l	F	25		
		30-d MATC	0.75–2.50 mg/l	F	14	pH	8.0
						HD	725
	Rainbow trout embryos-larvae	58-d MATC	<0.20 mg/l	F	14	pH	7.8
						HD	580
Phenol[268]	Bluegill embryos-larvae	3- to 4-d LC_{50}	2.42–3.34 mg/l	F	18–26	pH	7.9
						HD	51
	Goldfish embryos-larvae	4-d LC_{50}	0.84–1.22 mg/l	F	18–26		
	Rainbow trout embryos	23-d LC_{50}	0.33 mg/l	F	13–15		
	larvae	4-d LC_{50}	0.31 mg/l	F	13–15		
	Bluegill embryos-larvae	3- to 4-d LC_{50}	1.69–2.43 mg/l	F	18–26	pH	7.8
						HD	199

Table 1—(Continued)

Pollutant[Ref.]	Species		Results	Exposure type	Temp. (°C)	Test conditions	
	Goldfish						
	embryos-larvae	4-d LC_{50}	0.34–0.39 mg/l	F	18–26		
	Rainbow trout						
	embryos	23-d LC_{50}	0.07 mg/l	F	13–15		
	larvae	4-d LC_{50}	0.07 mg/l	F	13–15		
Phenyl mercuric acetate (formulation)[50]	Mosquitofish	96-h LC_{50}	44 µg/l	S	27	pH	7.5
						HD	32
Phenyl mercuric acetate (technical grade)[50]	Mosquitofish	96-h LC_{50}	37 µg/l	S	27	pH	7.5
						HD	32
Potassium permanganate[128]	American eel						
	black eel stage	96-h LC_{50}	4.86 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Potassium permanganate[129]	American eel						
	glass eel stage	96-h LC_{50}	3.06 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Potassium permanganate[130]	American eel						
	yellow phase	96-h LC_{50}	21.6 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Pro-Noxfish (EC 2.5% Rotenone, 2.2% Sulfoxide)[123]	Golden shiner	48-h LC_{50}	0.1–1.0 mg/l	S	15	pH	7.5–7.7
Propamocarb[118]	Channel catfish	28-d BCF	1.5–3.0	S	—		—
	Bluegill	28-d BCF	40–43	F	—		—
Propoxur[53]	Atlantic salmon	24-h LC_{50}	6.0 mg/l	S	8	pH	6.8
	Brook trout	24-h LC_{50}	11.2 mg/l	S	8		
Prudhoe Bay crude oil (water soluble fraction)[266]	Coho salmon						
	fry	96-h LC_{50}	10.38 mg/l	F	—		—
Resorcinol[262]	Rainbow trout	96-h LC_{50}	>100 mg/l	F	14	pH	8.0
	Fathead minnow	96-h LC_{50}	100 mg/l	F	14	HD	707
Rotenone (1% Derris Powder)[124]	Rainbow trout	96-h LC_{50}	350 µg/l	R	14	pH	7.4
						HD	78
Selenium[191]	Fathead minnow						
	fry	96-h LC_{50}	1.0 mg/l	F	25	pH	7.3
						HD	329
Selenium (selenite)[192]	Carp	96-h LC_{50}	35 mg/l	S	23–25	pH	7.2–8.2
Selenium (sodium selenite)[251]	Rainbow trout	96-h LC_{50}	8.1 mg/l	F	15	pH	7.9
		9-d LC_{50}	6.5 mg/l	F	15	HD	135
Sevin[56]	*Mystus vittatus*	72-h LC_{50}	17.5 mg/l	S	—		—

Sevin[27]	*Labeo rohita*	96-h LC_{50}	4.6–7.8 mg/l	F	28		—
Shell Dispersant LTX[28b]	Rainbow trout	96-h LC_{50}	750 mg/l	S	15	pH	6.1–7.1
						HD	12–24
Sodium chloride[128]	American eel black eel stage	96-h LC_{50}	21 450 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Sodium chloride[129]	American eel glass eel stage	96-h LC_{50}	17.9 mg/l	S	22	pH	7.2–7.6
						HD	40–48
Sodium pentachlorophenate[291]	Coho salmon juvenile	96-h LC_{50}	0.065 mg/l	F	13	pH	6.9–7.5
						HD	54
Sugee #2[288]	Rainbow trout	96-h LC_{50}	>1 000 mg/l	S	15	pH	6.1–7.1
						HD	12–24
Synperonic OSD 20[288]	Rainbow trout	96-h LC_{50}	80 mg/l	S	15	pH	6.1–7.1
						HD	12–24
2,4,5-T[113]	Coho salmon	96-h LC_{50}	>10 mg/l	S	10	pH	7.5
						HD	99
Tallow ethoxylate[266]	Harlequin fish	96-h LC_{50}	0.7 mg/l	R	20	HD	20
	Brown trout	96-h LC_{50}	0.4 mg/l	R	15	HD	26–30
	Golden orfe	96-h LC_{50}	2.3 mg/l	R	20	HD	268
		96-h LC_{50}	2.5 mg/l	S	20		
Terbutryne[109]	Grass carp	96-h LC_{50}	5.8 mg/l	F	18–21	pH	8.1
						HD	270
1,2,3,5-Tetrachlorobenzene[304]	Guppy	7-d BCF	72 000	F	21		—
TFM (35.7% active ingredient)[127]	Rainbow trout embryos (6- to 32-d-old)	24-h LC_{50}	4–43 mg/l	F	10	pH	7.6
						HD	354
Toluene[286]	Coho salmon fry	96-h LC_{50}	9.36 µl/l	F	—		—
Tordon 101[113]	Coho salmon	24-h LC_{50}	20 mg/l	S	10	pH	7.4–7.6
						HD	102
Tordon 22K[113]	Coho salmon	24-h LC_{50}	17.5 mg/l	S	10		—
Toxaphene[49]	Channel catfish fingerlings	96-h LC_{50}	2.7–7.5 µg/l	S	15–25	pH	6.5—8.3
						HD	272
	fingerlings	12- to 21-d LC_{50}	2.1–3.7 µg/l	F	20–25	pH	7.4
	Bluegill	96-h LC_{50}	3.4–4.7 µg/l	F	20–25	HD	272
		34-d LC_{50}	0.7 µg/l	F	20		

Table 1—(Continued)

Pollutant[Ref]	Species		Results	Exposure type	Temp. (°C)	Test conditions	
	Channel catfish						
	yolk-sac fry	96-h LC_{50}	8.0 µg/l	S	25	pH	7.4
	swim-up fry	96-h LC_{50}	0.8 µg/l	S	25	HD	40
	fingerlings	96-h LC_{50}	2.8 µg/l	S	25		
	Bluegill	96-h LC_{50}	2.4–2.6 µg/l	S	20–25		
Toxaphene (100% active ingredient)[49]	Fathead minnow	96-h LC_{50}	5–7 µg/l	F	20–25	pH	7.4
		16-d LC_{50}	1.5 µg/l	F	25	HD	272
		24-d LC_{50}	2.6 µg/l	F	20		
		96-h LC_{50}	20–23 µg/l	S	20–25	pH	7.4
						HD	40
1,2,3-Trichlorobenzene[304]	Guppy						
	female	2-d BCF	13 000	F	21		—
1,2,4-Trichlorobenzene[301]	Rainbow trout						
	fingerling muscle	8-h BCF	51	S	—		—
	fingerling muscle	35-d BCF	89	F	—		—
1,3,5-Trichlorobenzene[304]	Guppy						
	female	2-d BCF	14 000	F	21		—
Underground coal gasification condenser water[356]	Rainbow trout	96-h LC_{50}	0.1%	F	14	pH	8.0
	Fathead minnow	96-h LC_{50}	0.11%	F	14	HD	655
Versene 100[295]	Bluegill	96-h LC_{50}	1 030 mg/l	S	22	pH	7.6
						HD	103
Versene acid (EDTA)[295]	Bluegill	96-h LC_{50}	159 mg/l	S	22	pH	7.6
						HD	103
Versene AG (7.5% Cu)[295]	Bluegill	96-h LC_{50}	555 mg/l	S	22	pH	7.6
						HD	103
Versene AG (2.5% Mg)[295]	Bluegill	96-h LC_{50}	2 520 mg/l	S	22	pH	7.6
						HD	103
Versene AG (5% Mn)[295]	Bluegill	96-h LC_{50}	14 100 mg/l	S	22	pH	7.6
						HD	103
Versene AG (+Zn)[295]	Bluegill	96-h LC_{50}	685 mg/l	S	22	pH	7.6
						HD	103
Versene CA[295]	Bluegill	96-h LC_{50}	2 340 mg/l	S	22	pH	7.6
						HD	103
Versene (Fe-3 specific)[295]	Bluegill	96-h LC_{50}	3 092 mg/l	S	22	pH	7.6
						HD	103
Versene $(NH_4)_2$ EDTA[295]	Bluegill	96-h LC_{50}	2 340 mg/l	S	22	pH	7.6
						HD	103

Versene $(NH_4)_4$ EDTA[296]	Bluegill	96-h LC_{50}	705 mg/l	S	22	pH	7.6
						HD	103
Versene Powder[295]	Bluegill	96-h LC_{50}	486 mg/l	S	22	pH	7.6
						HD	103
Versenex 80[295]	Bluegill	96-h LC_{50}	1 115 mg/l	S	22	pH	7.6
						HD	103
Versenol AG (5% Fe)[295]	Bluegill	96-h LC_{50}	8 100 mg/l	S	22	pH	7.6
						HD	103
Versenol 120[296]	Bluegill	96-h LC_{50}	808 mg/l	S	22	pH	7.6
						HD	103
Waste oil[287]	Flagfish (*Jordanella floridae*) juvenile						
		96-h LC_{50} as water soluble fraction	36 200 µl/l	S	25	pH	7.1–7.7
						HD	42–49
		96-h LC_{50} as water soluble fraction	9 500 µl/l	F	25		
		96-h LC_{50} as emulsion	485 µl/l	S	25		
		96-h LC_{50} as emulsion	82.7 µl/l	F	25		
		~100-d MATC as water soluble fraction	325–930 µl/l	F	24	pH	7.2–7.8
						HD	43–48
Ziegler alcohol ethoxylate (C12-14, 8 EO)[266]	Brown trout	96-h LC_{50}	0.8 mg/l	R	15	HD	26–30
	Golden orfe	96-h LC_{50}	2.7 mg/l	R	20	HD	268
		96-h LC_{50}	1.8 mg/l	S	20		
Ziegler alcohol ethoxylate (C12-14, 10-11 EO)[266]	Harlequin fish	96-h LC_{50}	1.6–2.8 mg/l	R	20	HD	20
	Brown trout	96-h LC_{50}	1.8 mg/l	R	15	HD	250
	Golden orfe	96-h LC_{50}	0.8 mg/l	R	15	HD	26–30
		96-h LC_{50}	4.1 mg/l	R	20	HD	268
		96-h LC_{50}	4.5 mg/l	S	20		
Zinc[183]	Bluegill	96-h LC_{50}	3.0–3.6 mg/l	F	19–22	pH	6.8–7.5
						HD	21–59
Zinc[181]	Northern squawfish juvenile	96-h LC_{50}	3 500–3 700 µg/l	F	10–12	pH	7.1–7.5
						HD	20–30
Zinc sulfate[185]	Guppy	48-h LC_{50}	61.5 mg/dm^3	S	—		—
Zinc sulfate[182]	*Puntius conchonius*	96-h LC_{50}	33.3 mg/l	S	13–19	pH	7.5
						HD	310

[a] S = static bioassay
[b] HD = hardness, mg/l
[c] BCF = bioconcentration factor
[d] F = flow-through bioassay
[e] R = renewal bioassay
[f] MATC = maximum acceptable toxicant concentration

sured from four sampling sites and from three collection periods. Higher concentrations generally occurred at two impounded locations; variations because of sampling period were not found. A total of 45 water, sediment, and creek chub (*Semotilis atromaculatus*) samples from Black Creek (Ind.) were analyzed by Dudley and Karr[42] for seven pesticides and PCBs. Low levels of dieldrin (mean 0.023 μg/g) and DDE (mean 0.016 μg/g) were found in all 17 fish samples, but were not detected in water or sediment. PCB concentrations in fish were 5 times greater than were organochlorine pesticide concentrations. The herbicide 2,4,5-T was detected in water samples but atrazine, alachlor, carbofuran, and malathion were not detected in any samples. Johnston and Cheverie[43] described the repopulation of a coastal stream by brook trout (*Salvelinus fontinalis*) and rainbow trout (*Salmo gairdneri*) after an accidental discharge of endosulfan. A 905-m study section of North Brook (Prince Edward Is.) had an estimated 246 to 456 brook trout and 5 to 27 rainbow trout per 100 m of stream before all of the fish were killed in August 1975. Populations in June 1976 were estimated to be 135 brook trout and 10 rainbow trout per 100 m of stream. Most of the emigrants were age I and age II fish from areas unaffected by the endosulfan. Reproduction in the study area was insignificant during the first year after the fish kill. Preliminary observations on the immediate effect of Abate on fish catches in the River Oti (Ghana) were made by Abban and Samman.[44] The concentration of Abate used for control of *Simulium damnosum* was between 0.05 and 0.10 mg for 10 min over *Simulium* breeding sites. Greater numbers of fish were caught in gill nets in the 24 h prior to treatment than in the 24 h after spraying. Within 5 min of Abate treatment, fish had erratic overt movements, but there was virtually no fish kill. Colwell and Schaefer[45] measured diflubenzuron residues in black crappie (*Pomoxis nigromaculatus*) and brown bullhead (*Ictalurus nebulosus*) under field treatment conditions, determined the effects on invertebrates, and correlated these findings with effects on these two species of fish. Young-of-the-y black crappie and brown bullheads accumulated diflubenzuron to a maximum of 466 and 388 ng/g, respectively, after exposure to a mean concentration of 13 μg/l for 14 d. By Day 7 after treatment, the chemical was not detected in fish. For 1 month following treatment, stomach content analyses indicated major alterations in diets of fish. However, growth rates and condition factors of the fish 3 months after treatment were similar to control fish from four untreated ponds. Residue levels of DDT, DDE, TDE, and ΣDDT in northern pike (*Esox lucius*) from the Richelieu River (Que.) were measured by Boileau *et al.*[46] The fish were netted between June 1974 and June 1975. Two years after the banning of DDT, residue levels of ΣDDT ranged from 0.2 μg/g in 2-year-old pike. Residues increased with age, and significant seasonal variations in the ΣDDT levels were found in 5- and 6-year-old pike. Pesticide levels in water, mud, zooplankton, and muscle and gill tissue from live and dead fish from Sathiar Reservoir (India) were measured by Kannan and Job.[47] Endrin, parathion, BHC, DDT, and DDE were detected at ng/g levels whereas only endrin and parathion were detected in water. Muscle from dead fish contained higher residue levels of BHC than muscle from live fish. Dean *et al.*[48] summarized research between 1962 and 1974 on lake trout (*Salvelinus namaycush*) reproduction in Lake George (N. Y.). DDT levels in females were still high, especially in older, larger fish. There was a good correlation between DDT levels in the eggs and in the muscle of the same fish.

Johnson and Julin[49] investigated the effect of some variables on the acute toxicity of toxaphene to fathead minnows (*Pimephales promelas*), channel catfish (*Ictalurus punctatus*), and bluegills (*Lepomis macrochirus*) (Table 1). In static tests at 20°C, 96-h LC_{50} values ranged from 2.6 μg/l for the bluegill to 20 μg/l for the fathead minnow. The 96-h LC_{50} values in flow-through tests ranged from 4.7 μg/l for the bluegill to 7.0 μg/l for the fathead minnow. Temperature, pH, and water hardness had only a minor influence on toxicity. Swim-up fry of channel catfish were more sensitive than yolk-sac fry or fingerlings. The acute toxicity of three organochlorine pesticides and some heavy metals to the mosquitofish (*Gambusia affinis*) was determined by Joshi and Rege[50] (Table 1). The 96-h LC_{50} values of the pesticides ranged from 6 μg/l for an emulsible concentrate of endosulfan to 2 900 μg/l for BHC. All the toxicants produced abortions in gravid females. The relative toxicity of dichlorvos to three size groups of *Tilapia mossambica* was evaluated by Rath and Misra.[51] The 96-h LC_{50} values ranged from 1.4 to 1.9 mg/l with the smaller fish being more sensitive (Table 1). Sahib and Rao[52] conducted two static acute toxicity tests using two series of concentrations to determine 48-h LC_{50} values of malathion to *Tilapia mossambica*. The mean 48-h LC_{50} value was 5.6 mg/l. Colquhoun and Dean[53] conducted static bioassays to produce estimates of the acute toxicity of propoxur (a carbamate insecticide) to brook trout and Atlantic (landlocked) salmon (*Salmo salar*). The 24-h LC_{50} value was 6.0 mg/l for brook trout and 11.2 mg/l for Atlantic salmon. Data on acute toxicity of aminocarb and components of the aminocarb formulation to Atlantic salmon, a freshwater clam, and five marine invertebrates were presented by McLeese *et al.*[54] The 96-h LC_{50} values of aminocarb, the formulation, and nonylphenol for juvenile Atlantic salmon were 8.7, 3.5, and 0.9 mg/l, respectively, indicating that the toxicity of the aminocarb formulation (Matacil) to the salmon was mainly because of nonylphenol. The acute toxicity of sevin and 1-naphthol, a metabolite of sevin, to two size groups of *Labeo rohita* was tested.[55] The acute toxicity of thiotox, dichlorvos, carbofuran, and various combinations of these three pesticides to *Mystus vittatus* was evaluated.[56] Most of the combinations of the three pesticides were synergistic. A combination of pesticides was synergistic or additive depending on relative concentrations used. Among the paired mixtures, the effects were mostly additive.

Yasuno *et al.*[57] examined the effects of temephos and fenitrothion on reproduction of the guppy (*Poecilia reticulata*). Temephos, at concentrations of 1.5, 1.0, 0.5, and 0.25 mg/l, did not produce an effect on the total number of embryos. However, the number of live larvae produced at 1.5 mg/l clearly decreased 1 month after the exposure and the effect continued during the 2-month, post-exposure period. The survival rates of young larvae reared for 2 weeks after birth during the exposure period did not differ for the various concentrations and control. Fenitrothion decreased the number of young during the second month of exposure at 1.0, 0.5, and 0.25 mg/l but not at 0.1 mg/l. A high rate of abortions was observed at 1.0, 0.5, and 0.25 mg/l during the post-exposure period. The weight of larvae at birth, before and after exposure, did not differ from control at 0.25 and 0.1 mg/l but decreased at

0.5 mg/l in the post-exposure months. The effects of sevin on survival, behavior, food intake, growth, and food conversion efficiency of the catfish, *Mystus vittatus*, were studied by Arunachalam *et al.*[58] The 72-h LC_{50} value was 17.5 mg/l and concentrations ranging from 5 to 12.5 mg/l accelerated swimming activity and increased frequency of opercular rate. A 27-d exposure to the same concentrations led to decreased feeding and growth rates. The toxicity and bioaccumulation of methoxychlor in aquatic invertebrates and of endrin in aquatic insects and black bullhead (*Ictalurus melas*) were determined in flow-through exposures by Anderson and DeFoe.[59] After a 28-d exposure, caddisfly was 3 times more sensitive than bullhead to endrin. The 96-h LC_{50} value of endrin for fish was 0.34 μg/l and toxicity increased with increased exposure time. The BCFs for the two exposures were 3 700 and 6 200, respectively.

The uptake and excretion of mirex and four Dechloranes (flame retardants) from water and from food by Atlantic salmon were compared by Zitko.[60] Only mirex and Dechlorane 602 were accumulated by fish from water. The 96-h BCF was 84 for mirex and 7.2 for Dechlorane 602. All compounds, including Dechloranes 603, 604, and Plus 25, were accumulated when administered in food. An impurity in Dechlorane 604 was accumulated to a larger extent than was Dechlorane 604. The half-lives of mirex and all Dechloranes ranged from 58 to 76 d. Herzberg *et al.*[61] measured the residues of lindane in the gills, head, and whole body of *Sarotherodon galilaeus* after exposure to various concentrations of the pesticide. The residues found in brain and heart of intoxicated fish did not differ significantly from those of controls. Lindane in the gills of intoxicated fish ranged from 0.33 to 0.48 μg/g—about the highest level before death. The values dropped to 0.26 to 0.40 μg/g upon recuperation when the fish were returned to clean water. The uptake and distribution of endrin in insecticide-susceptible and insecticide-resistant mosquitofish were measured by Hunsinger *et al.*[62] Fish were exposed to equal (10.2 μg/l) and equitoxic (0.6 μg/l for susceptible fish and 314 μg/l for resistant fish) levels of ^{14}C-endrin for up to 8 h. At all exposures, differences in uptake and distribution of ^{14}C-endrin were found between the two fish populations. At an 8-h equitoxic exposure there was 72 times as much ^{14}C-endrin in the brains of resistant fish as in the brains of sensitive fish. Gall bladder/liver ratios indicated that resistant fish could effectively excrete endrin over a wider range of exposures than sensitive fish. Skea *et al.*[63] measured effects of trimming and cooking brown trout (*Salmo trutta*) and smallmouth bass (*Micropterus dolomieui*) on residue levels of mirex, Aroclor 1254, and DDE. Proper trimming reduced levels of all three organochlorine compounds by 43 to 64%. Of the cooking methods (smoking, baking, broiling, and deep frying), only deep frying of smallmouth bass produced consistently high reduction of all three compounds. The uptake, transport, and accumulation of lindane in a food chain consisting of *Chlorella* sp., *Daphnia magna*, and threespine stickleback (*Gasterosteus aculeatus*) was measured by Hansen.[64] In fish exposed for 9 days, lindane was taken up in proportion to its concentration in water at concentrations between 10 and 70 μg/l. It was observed that female sticklebacks with their higher fat content accumulated more lindane than males. Males in breeding condition accumulated 5.8 times as much lindane as those in a normal condition indicating that the rate of metabolic activity was important. The results showed that the uptake from water was rapid whereas the uptake from food was slow and depended on the duration of exposure and the feeding rate. Ellgehausen *et al.*[65] conducted studies with alga (*Scenedesmus acutus*), daphnids (*Daphnia magna*), and black bullheads to determine the kinetics of bioaccumulation and depuration of 10 pesticides and to estimate the contribution of food chain transfer of pesticide residues to total bioaccumulation. It was shown that bioaccumulation of nonionic organic chemicals from water could be described as sorption or as a distribution phenomenon that was correlated with the n-octanol/water partition coefficient of the compound. Depuration of the pesticides from aquatic organisms followed second-order kinetics. Half-lives, as well as the reciprocals of the rate constants of depuration, were found to be highly correlated with the lipophilicity of the pesticide. Transfer of pesticide residues through the food chain was found to be of less importance compared with direct uptake from water.

The uptake and metabolism of chlordane by cutthroat trout (*Salmo clarki*) and California newts (*Tarichia torosa*) from a lake treated with 10 μg/l of technical chlordane was measured by Albright *et al.*[66] At Days 93, 279, 421, and 1 014, concentrations of total technical chlordane in the trout (except livers) were 36.1, 4.0, 2.2, and 0.1 μg/g, respectively. The heptachlor was quantitatively epoxidized in newts after 14 days, but some heptachlor was still present in the fish after 93 days. Heptachlor was not detected in either species after 279 days. Of the seven individually determined constituents of technical chlordane, trans-nonachlor was by far the most persistent. The results indicated that newt metabolized and eliminated chlordane residues more effectively than fish. In another study,[67] the metabolism, distribution, and elimination of cis[^{14}C]chlordane in the cichlid (*Cichlasoma* sp.) was measured. Fish were exposed to 80 μg/l of cis[^{14}C]chlordane for 72 h and some returned to clean water. The fish accumulated 2.8, 1.2, and 1.1 mg/kg of the compound in visceral fat, bile, and gall bladder, respectively. At the end of the 72-h exposure, recovery of radioactivity was estimated to be 64.8 and 6.1% in fish and water, respectively. The rate of elimination of the radioactivity was about 2.9%/week. Dichlorochlordene, oxychlordane, chlordene chlorohydrin, dihydroxyheptachlor, dihydroxydihydrochlordene plus four unidentified compounds accounted for 12.5% of the radiocarbon recovered from fish and exposure water. The remainder was unchanged cis-chlordane. The same authors[68] measured cis[^{14}C]chlordane in goldfish (*Carassius auratus*). Fish were exposed to 26 μg/l for 24 h and returned to clean water. Over 99% of the radioactivity recovered from the fish after 10 and 25 d in clean water was unchanged cis-chlordane. Hematological alterations of singii (*Saccobranchus fossilis*) exposed to 120 μg/l of chlordane for 60 d were examined by Verma *et al.*[69] Fish were sampled at 15-d intervals. Hemoglobin percentage, RBC, WBC, and hematocrit value increased whereas erythrocyte sedimentation rate and clotting time decreased. Of the 12 blood constituents measured, nine increased and proteins and cholesterol decreased. In related studies, the sensitivity of ATPase systems from brain, gill, liver, and kidney of *Labeo rohita* and *Saccobranchus fossilis* and *in vitro* treatment with chlordane were measured.[70] Inhibition of the ATPase systems in all tissues of *Labeo rohita* was greater than in *Saccobranchus fossilis*. For both species, inhibition was greatest in brain followed by gill,

kidney, and liver, and inhibition was increased with increased concentrations. Bansal *et al.*[71] measured effects of chlordane on blood chemistry of *Labeo rohita* exposed to 17, 23, 35, and 70 μg/l for 60 days. Of the organic components and serum electrolytes measured, there was no effect on protein, lactate, NPN, and K, whereas there was a statistically significant ($P = 0.05$) effect on 10 parameters at one or more concentrations. The absorption, metabolism, and elimination of ^{14}C-photodieldrin from bluegills exposed to 20 μg/l for 48 h were measured by Sudershan and Khan.[72] Absorption was rapid with about 95% of the activity of the water being lost in 20 h. The fish eliminated 50% of the absorbed radioactivity in 3 weeks. About 27% of the eliminated radioactivity was unaltered photodieldrin whereas the remainder contained at least 11 metabolites in free and conjugated forms. Photodieldrin ketone was the major excretory product. Solvent extracts of the whole fish contained four metabolites in addition to about 90% of the unmetabolized photodieldrin. The ketone derivative was the most abundant of the metabolic products. Goldfish exposed to 20 μg/l for 48 h to ^{14}C-photodieldrin in a static system absorbed 80% of the radioactivity within 20 h.[73] The absorbed radioactivity had a half-life of 3 weeks. Photodieldrin and a ketone derivative accounted for 59 and 10.4%, respectively, of the eliminated radioactivity. Approximately 15 other polar and nonpolar metabolites were found in the water. Photodieldrin and the ketone derivative accounted for 77 and 5.4%, respectively, of the residues in the fish and the radioactivity in fish contained 13 metabolites. Elimination and metabolism studies of ^{14}C-heptachlor showed that goldfish injected in the body cavity with 38.2 μg of the compound eliminated about 18% of the dose in 10 d.[74] Analysis of the fish after 10 d revealed the presence of five compounds with 91.2% of unchanged heptachlor. The other compounds were heptachlor epoxide (5.4%), 1-hydroxychlordene (1%), 1-hydroxy-2,3-epoxychlordene (1.1%), and a conjugate (1.2%). Analysis of water and feces gave only polar products with the conjugate being the most abundant.

Chambers and Yarbrough[75] made a seasonal study of microsomal mixed-function oxidase components in (organochlorine) insecticide resistant and susceptible mosquitofish. Most of the parameters had seasonal cycles with the highest values occurring during the late fall and winter. The range of concentrations or specific activities of most of the components was about the same for both populations. However, resistant fish had relatively larger livers with greater amounts of the microsomal components. The activity of succinic dehydrogenase, pyruvic dehydrogenase, and lactic dehydrogenase of the kidney and liver of *Ophiocephalus punctatus* exposed to endrin at the 96-h LC_{50} concentration of 33 μg/l was examined by Sharma *et al.*[76] Only the activity of lactic dehydrogenase of the kidney exposed to endrin was significantly different ($P = 0.05$) than that of control fish. McCorkle *et al.*[77] measured enzyme activities after exposing channel catfish to 1, 100, 200, and 400 mg/kg of dietary mirex. Specific activities of lactic dehydrogenase, malic dehydrogenase, glutamate pyruvate transaminase, and glutamate oxaloacetate transaminase were determined in brain, gill, liver, and muscle at 1, 2, 3, and 4 weeks of exposure and few changes in enzyme specific activities were found. Leatherland and Sonstegard[78] assessed the effect of dietary mirex and PCB (Aroclor 1254), fed at the rate of 500 μg/g dry food, on the thyroid activity and lipid reserves in rainbow trout. There was no effect of PCB on thyroid histology or on serum thyroid hormone levels but liver weights and total-liver lipid content were significantly greater in PCB-fed fish as compared to control fish. Mirex caused significant reductions in serum T_4 and T_3 levels without apparent changes in the histological appearance of the thyroid. Mirex had no apparent effect on liver size or lipid content, but there was a significant decline in carcass lipid content which persisted for 25 d after return of fish to the control diet. In a similar study,[79] trout fed high mirex (50 mg/kg dry feed) or high PCB (500 mg/kg dry feed) levels had significantly lower serum thyroid hormone levels. Trout fed low mirex (5 mg/kg) or low PCB (50 mg/kg) levels had thyroid hormone levels similar to controls. Trout fed the mixed diet, PCBs (50 mg/kg) plus mirex (5 mg/kg), had significantly greater triiodothyronine levels after starvation and subsequent testosterone administration. These fish also had whole-body PCB levels 60% of those fed PCBs alone (50 mg/kg), but contained similar levels of mirex to those fed mirex alone (5 mg/kg). The hepatosomatic index (HSI) in fish fed PCBs was significantly greater than the controls in the fed groups. However, after 14 days of food deprivation and subsequent testosterone administration, the HSI was no different from controls.

Duangsawasdi and Klaverkamp[80] investigated the acute toxicity of acephate and fenitrothion to rainbow trout. The 24-h LC_{50} value for acephate at 22°C was significantly less than the values at 8 and 15°C. In the fenitrothion exposure, all three 24-h LC_{50} values were significantly different, being more toxic at higher temperatures. For both organophosphate compounds, however, the 96-h LC_{50} values at 8, 15, and 22°C were similar. Both insecticides produced a decrease in heart rate and an increase in ventilation rate and buccal amplitude. Fenitrothion produced an increase in cough frequency, but acephate did not. For adult trout exposed to the approximate 48-h LC_{50} concentration of 2 000 mg acephate/l or 2 mg fenitrothion/l, cholinesterase activity of the skeletal muscle after a 24-h exposure was approximately the same. However the cholinesterase activity of brain, gill, heart, RBC, and serum was considerably lower in the fenitrothion-exposed fish. A similar study was made to investigate the brain acetylcholinesterase (AChE) inhibition and biotransformation by liver homogenate to explain the temperature-dependent acute toxicity of fenitrothion in rainbow trout and to explain the three orders of magnitude difference in the 96-h LC_{50} values of fenitrothion and acephate to this species.[81] Concentrations of both organophosphate insecticides required to produce 50% inhibition of brain AChE were similar, whereas fenitrooxon, the oxidative desulfuration metabolite of fenitrothion, was approximately five orders of magnitude more potent. Incubation with liver homogenates demonstrated that a more potent brain AChE inhibitor was produced from acephate but not from fenitrothion. It was concluded that hepatic biotransformation of fenitrothion to fenitrooxon does not explain the temperature-dependent toxicity of fenitrothion or the difference in acute toxicity of the two pesticides. Adeney and Matthiessen[82] exposed *Sarotherodon mossambicus* to concentrations of Abate greater than that used for control of blackfly larvae in West African rivers. Growth of fish exposed to 0.05 mg/l for 16 weeks and AChE activity, plasma glucose, hemoglobin, and plasma sodium and potassium of fish exposed to 0.1 mg/l for 16 d was not significantly different from that of control fish.

Some biochemical changes in the air-breathing catfish (*Clarias batrachus*) exposed to 500 μg/l of malathion for 40 d were examined by Mukhopadhyay and Dehadrai.[83] Incorporation of l-lysine-U-^{14}C into protein of liver was significantly reduced and could not be recoupled even with higher dietary protein. Hematological studies indicated that malathion caused a decrease in erythrocyte count and an increase in the total leucocyte count. In addition, eosinophil and neutrophil leucocytes were increased with no significant change in the non-granular varieties. There was no effect on hemoglobin, hematocrit, or erythrocyte sedimentation rate. Malathion-exposed fish had little change in hepatic protein and nucleic acids but had a marked decrease in liver glycogen and a significant increase in serum glucose and free amino acid. Sahib and Rao[84] exposed *Tilapia mossambica* to 2 mg/l of malathion for 48 h and measured acetylcholinesterase activity in brain, muscle, gill, and liver tissue. There was a significant inhibition of the activity in all four tissues. Inhibition in brain, muscle, gill, and liver was 48, 46, 39, and 35%, respectively. Esterine tested on AChE of normal tissue at 0.03 mM in brain, 0.026 mM in muscle, 0.02 mM in gill, and 0.016 mM in liver induced the same percentage of inhibition of AChE as in the tissue of fish exposed to 2 mg/l of malathion. The effects of a sublethal exposure to a concentration of 0.1 mg/l of methyl parathion for 48 h on two oxidative enzymes and four organic constituents of muscle, gill, and liver of Mozambique mouthbrooder was measured by Rao and Rao.[85] There were significant effects on all six components of all three tissues. Protein and free amino acid content increased whereas levels of succinate dehydrogenase and lactate dehydrogenase enzyme activities decreased, and carbohydrate and glycogen contents decreased. *Ophiocephalus punctatus* were exposed to diazinon at the concentration of 3.1 mg/l for 96 h and to 0.31 mg/l for 15 and 30 d.[86] At all three exposures, there was a significant decrease in the activity of acid phosphatase, glucose-6-phosphate, lactic dehydrogenase, pyruvic dehydrogenase, succinic dehydrogenase, and cholinesterase. Alkaline phosphatase activity was reduced at 96-h exposure and increased at 30-d exposure. Adenosine triphosphatase activity was decreased only at the 96-h and 15-d exposure.

The effects of exposure to a lethal concentration (6 mg/l) of sumithion on carbohydrate metabolism in Mozambique mouthbrooder were examined by Koundinya and Ramamurthi.[87] The exposure to 6 mg/l increased blood glucose levels and phosphorylase activity, but hepatic glycogen decreased. Hematological values of *Sarotherodon mossambicus* exposed for 30 d to a sublethal concentration of 6 mg/l of sumithion and 4 mg/l of sevin were described.[88] Red blood cells, packed cell volume, and hemoglobin were significantly increased in the exposed fish. There was no significant effect of either pesticide on mean corpuscular volume, mean corpuscular hemoglobin, or mean corpuscular hemoglobin concentration. *In vitro* metabolism of carbaryl by liver explants of bluegill, channel catfish, yellow perch (*Perca flavescens*), goldfish, and kissing gourami (*Heolstoma temmincki*) was examined by Chin *et al.*[89] Based on metabolic profile analysis, livers from each species performed the metabolic processes of demethylation and/or hydrolysis, hydroxylation, and oxidation, followed by conjugation. The most significant metabolite in all five species chromatographed as a dihydrodihydroxycarbaryl glucuronide. The effects of Cythion (malathion formulation) and Hexadrin (endrin formulation) on thyroid activity and thyrotrophic potency of the pituitary gland and blood serum of *Heteropneustes fossilis* were studied by Singh and Singh.[90] After a 4-wk exposure to 8 and 0.6 μg/l of Hexadrin and 35 mg/l of Cythion, there was reduced thyroid activity and reduced pituitary and serum thyroid-stimulating hormone (TSH) content. At a concentration of 0.09 mg/l of Cythion, pituitary TSH levels were not affected but serum TSH levels and thyroid activity were reduced. These authors[91] also analyzed the effects of these pesticides on the reproductive cycle of *Heteropneustes fossilis*. Neither pesticide at sublethal concentrations caused significant changes in the lipid concentration in the ovary but significantly decreased liver lipid during the preparatory and late post-spawning phases. Ovarian and serum cholesterol were both increased during these phases, whereas liver cholesterol was unaffected. Studies on the effects of Cythion and Hexadrin on gravid females of this species[92] showed that a lethal and sublethal concentration of both pesticides reduced ovarian ^{32}P uptake and also reduced the level of total gonadotrophin in the pituitary gland and serum. Administration of luteinizing hormone or pituitary extract during the exposure of fish to a lethal and sublethal concentration of malathion and a sublethal concentration of endrin increased ovarian ^{32}P uptake. The acute effects of these pesticides on lipid and cholesterol content of liver, ovary, and blood serum of female *Heteropneustes fossilis* were also measured.[93] Exposures to lethal and sublethal concentrations of both pesticides for 96 h caused a significant increase in ovarian cholesterol but not in blood serum and liver cholesterol. These exposures also did not affect liver and ovarian lipid content. *In vitro* effects of chlordane and Metasystox dissolved in various organic solvents on the inhibition of ATPase of gills and brain of *Labeo rohita* were examined by Dalela *et al.*[94] The percent inhibition of ATPase activity of 0.25 μM of chlordane or 5.0 μM of Metasystox varied with the solvent used, but the variation was greater with chlordane. In both tissues, effects of these two pesticides were greater on Na^+, K^+ ATPase than on Mg^{++} ATPase activity. The percent inhibition of ATPase in the brain was greater than that of the gill.

Oxygen consumption of rohu (*Labeo rohita*) exposed for 96 h to lethal and sublethal concentrations of chlordane, Metasystox, and sevin was measured by Bansal *et al.*[95] At the 96-h LC_{50} concentration, an all or nothing response was observed. At sublethal concentrations, oxygen consumption was in three phases: sensitization, responsive, and normalization. Oxygen consumption data showed that there was a critical threshold time for the responsive phase, between 6 and 24 h for chlordane, from the beginning up to 6 h for Metasystox, and between 2 and 24 h for sevin. Wroblewski[96] determined the acute toxicity of chlorfenvinphos to the guppy. The 96-h LC_{50} values for small guppies (8 mg) was ~0.9 mg/l and for larger fish (32 mg) was 0.6 mg/l. Five behavioral symptoms of intoxication were described. When intoxicated fish were returned to clean water, the first three symptoms receded in opposite sequence, whereas the two symptoms of more severe intoxication were irreversible. Food consumption, respiration, and growth also were determined in fish returned to clean water and changes in food consumption proved to be the best index of poisoning. Ellgaard *et al.*[97] measured the locomotor activity of mosquitofish and goldfish exposed to 0.2 mg/l of methoprene and the locomotor activity of mosquitofish exposed to 0.2 mg/

l of diflubenzuron. This concentration of the two insect growth regulators was approximately 10 times greater than that generally recommended for application. Exposure to mosquitofish for 14 d and goldfish for 13 d did not significantly change the locomotor activity of either species. Within 2 d mosquitofish exposed to diflubenzuron were 2.5 times more active than control fish. Maximum activity was observed on Days 4 through 8 when they were 4 times as active as controls. Activity decreased to control level by Day 14. Narain and Srivastava[98] examined the blood cells of the Indian catfish (*Heteropneustes fossilis*) exposed to fertilizers, BHC, endrin, monochrotophos, phosphamidon, and wastewater. Structural abnormalities found were: lysis, poikilocytosis, denucleation, cytoplasmic vacuolization, cytoplasmic warping, cytoplasmic inclusions in erythrocytes, vacuolization in agranulocytic leucocytes, and appearance of smudges. The relative toxicity of endosulfan, two of its isomers, and formulated products to *Labeo rohita* was measured by Rao *et al.*[99] The 96-h LC_{50} values ranged from 0.33 μg/l for endosulfan A to 7.1 μg/l for endosulfan B. Endosulfan A was 3.3 times more toxic than technical grade endosulfan. Endosulfan and its metabolites were measured qualitatively in seven tissues of fish exposed to 4 μg/l for 96 h. The main method of detoxification was by formation of endosulfan ether in the liver. Both lethal and sublethal concentrations of the pesticide increased oxygen consumption. Histological studies indicated only minor changes in the liver. Histological damage to the gills of *Channa gachua* from acute and subacute exposure of emulsible concentrates of endosulfan and Rogor was observed by Dalela *et al.*[100] Lethal exposure to 12 μg/l of endosulfan caused separation of respiratory gill epithelium from the basement membrane, pronounced hyperemia, necrosis, fusion of adjacent gill lamellae, erosion at the distal end of gill filaments, and loss of cell membrane. Lethal exposure to 6.5 mg/l of Rogor also caused erosion at the distal end of gill filaments and loss of cell membrane. Sublethal exposure to 4 μg/l of endosulfan or 1.5 mg/l of Rogor caused increasing damage to gills with increased times of exposure.

Herbicides. The effects of impounding the Des Moines River (Iowa) on the pesticide concentrations in warmwater fishes were evaluated by Leung.[101] Atrazine, alachlor, and bladex concentrations fluctuated seasonally in the Des Moines River from September 1977 through November 1978. These herbicides were not detected in seven species of fish collected above, within, or below the Saylorville Reservoir. Dieldrin, heptachlor-epoxide, p,p'-DDT, p,p'-DDE, and p,p'-DDD were detected in most fish samples. There were no seasonal patterns in fish residues. Classical biomagnification and spatial trends in residues of these insecticides were not noticeable in the fish. Klaassen and Kadoum[102] examined the distribution and retention of atrazine and carbofuran in three farm ponds. The first pond received an application of 0.3 mg/l of atrazine for two yearly treatments. The second pond received an application of 0.025 mg/l of carbofuran the first year and 0.05 mg/l for the second year. The third pond received an application of 0.3 mg/l of atrazine plus 0.025 mg/l of carbofuran the first year and 0.3 mg/l of atrazine plus 0.05 mg/l of carbofuran the second year. Carbofuran was found only in water and mud immediately after application. It was not detected in zooplankton, black bullheads, or bluegills. Soon after atrazine was applied it was found in water, mud, and animals, including the bluegill. However, residue levels in all animals were similar to water concentrations and no adverse effects on the animals were detected.

Arnold[103] conducted field trials to evaluate the efficacy of fluridone, a new aquatic herbicide, on aquatic plants and its effect on phytoplankton, zooplankton, benthic organisms, and fish. No adverse effects were observed on bluegills, bass, or catfish. Fluridone was found in bluegills, but only at low levels. The dissipation of fluridone from lakes and ponds was determined by West *et al.*[104] Fluridone had a half-life that averaged 5 d and varied from 1 to 11 d. The herbicide had a very low potential for bioconcentration in aquatic plants, zooplankton, and fish. As a general rule, fluridone was not detected in fish when it was no longer detected in water. The concentration of fluridone in fish was always less than 2 times the concentration in water. The effects of the herbicide 2,4-D on the ecology of experimental ponds was assessed by Boyle.[105] The mean length, weight, and condition factor of bluegills taken from the four high treatment ponds (1.0 mg/l) were significantly greater than those in the four low treatment (0.5 mg/l) or control ponds. Survival did not appear to vary among treatments. It was concluded that stimulation of phytoplankton productivity may be reflected in increased growth of fishes, although the possibility exists that 2,4-D may act as a growth stimulus in a more direct way. Dodson and Mayfield[106] observed the effect of the butoxyethanol ester of 2,4-D and its commercial formulation Aqua-Kleen on the rheotropic response of rainbow trout. Toxicological modification of rheotropism occurred that would lead in nature to an increased incidence of downstream movement. The ester was taken up rapidly by fish, hydrolyzed to 2,4-D, and secreted into the water. Skea and Simonin[107] used fathead minnows and largemouth bass (*Micropterus salmoides*) to determine the acute toxicity of Cutrine, a chelated copper algicide. In addition, its effects on filamentous algae and the two fish species were determined under field conditions. The 96-h LC_{50} values of Cutrine as copper, was 6.4 mg/l for bass and 0.21 and 0.12 mg/l for the minnow at 18 and 14°C, respectively. When Cutrine was applied at the recommended rate to half of a fathead minnow rearing pond, the amount of filamentous algae was reduced and mortality of largemouth bass and fathead minnows caged in the treated area of the pond was less than 10%. Dodson and Mayfield[108] observed the rheotropic response of rainbow trout in response to a simulated water current following 24-h exposures to three field application concentrations of diquat and simazine. Modification of rheotaxis and swimming speeds were observed in trout exposed to diquat and its formulation Reglone A resulting in an increased incidence of downstream drift. Fish exposed to simazine and Tween 80 showed significant variation in their behavior pattern; however, no such variation was observed in fish exposed to Princep 80W, a wettable powder formulation of simazine. Residues in fish after the rheotropic response study were measured and simazine residues increased in proportion to treatment concentration whereas diquat residues leveled off with increasing treatment concentrations.

The acute toxicity of 10 herbicides to grass carp (*Ctenophayngodon idella*) was measured by Tooby *et al.*[109] (Table 1). All except cyanatryn and diuron have been cleared for use under the Pesticide Safety Precautions Scheme of the Ministry of Agriculture, Fisheries and Food (Great Britain). Except for asulam and dalapon, for which mortality data were not ob-

tained, the concentration-response relationship for each pesticide did not reach an asymptotic concentration during the 5-d exposure period. A preliminary evaluation of the risk to this species indicated that when these herbicides were used at the recommended rate, the maximum concentration likely to be found in water would cause little harm to the fish. Upadhyaya and Rao[110] exposed three fish species (*Puntius ticto, Tilapia mossambica*, and *Heteropneustes fossilis*) to 1, 5, and 10 mg/l of tafazine for 36 d. In general, the opercular rate increased with increased concentration. Acute toxicity tests were conducted by McCann and Hitch[111] in an effort to resolve the difference in reported 96-h LC_{50} values of simazine to striped bass (*Morone saxatilis*). In both soft and hard water the 96-h LC_{50} values of Aquazine (simazine—80% wettable powder) was >180 mg/l. Shehata and Nawar[112] determined the toxicity of the cationic polyelectrolyte, Anti-Germ 50 (an algicide), to algae and to the Nile fish (*Tilapia nilotica*) in dechlorinated tap water and in Nile River water. The 96-h LC_{50} for fish in Nile water (8 mg/l) was twice that in dechlorinated tap water, presumed to be because of the presence of suspended solids in the river water. Lorz *et al.*[113] examined some effects of 12 herbicides on yearling coho salmon (*Oncorhynchus kisutch*). Acrolein and dinoseb were the most toxic of the herbicides, having 96-h LC_{50} values of 68 and 100 μg/l, respectively (Table 1). Fish exposed to Amitrol-T, diquat, and paraquat in freshwater exhibited concentration-dependent effects in subsequent seawater entry tests. No effect on the (Na, K)-stimulated ATPase activity of the gills was found with any of the herbicides. The effect of sublethal concentration of Tordon 101, dinoseb, and diquat on migration was tested by releasing exposed fish into a natural stream. Only diquat produced a significant reduction in downstream migration.

The uptake and elimination of ^{14}C-fosamine ammonium by channel catfish was determined by Han.[114] Catfish were exposed for 4 weeks to 1.1 mg/l of the herbicide and were also exposed in 140 l of water containing 10 kg of soil treated with 151 mg of ^{14}C-fosamine ammonium that had been aged for 30 days prior to being added to the water. In both exposures, the residue levels reached plateau values in 2 to 3 weeks with a BCF of <1. In 2-wk depuration periods, residue levels dropped 50 to 90%. Folmar[115] conducted studies to determine if short-term application of acrolein and 2,4-D at field application concentrations would produce an offensive flavor in the flesh of rainbow trout. The data from the taste panel indicated that experimental concentrations of both acrolein (0.1 mg/l) and 2,4-D (0.5 mg/l) applied for 4 h, and the fish collected 1, 4, and 7 d post-treatment of 2,4-D and 1 and 4 d post-treatment of acrolein tainted the flesh of rainbow trout to make them unpalatable. Tissue residue analysis of the 2,4-D samples showed no significant accumulation of the parent compound. The behavior and fate of 4-chloro-2-methylphenoxyacetic acid (MCPA) and 4,6-dichloro-o-cresol in an aquatic-terrestrial model ecosystem was studied by Virtanen *et al.*[116] The rate of treatment simulated the usage of these two herbicides. MCPA was found in only one of six goldfish, and the BCF for this fish was <1. None of the components of the model contained measurable amounts (10 ng) of 4,6-dichloro-o-cresol. Isensee and Yockim[117] compared a recirculating static model ecosystem with a flow-through model ecosystem to test the behavior of pentachloronitrobenzene (a fungicide) and two herbicides, simazine and trifluralin, in aquatic model ecosystems containing *Oedogonium cardoaci, Helisoma* sp., *Daphnia magna* and mosquitofish. The total amount of ^{14}C-labeled pentachloronitrobenzene accumulated by all organisms was about the same in each of the two ecosystems at similar treatment rates. Residues in the organisms reached equilibrium levels with water in 3 to 7 days and decreased 50 to 95% when returned to clean water for 10 days. Simazine (^{14}C-labeled) accumulation by snails and mosquitofish was similar between systems at similar treatment rates, whereas algae and daphnids accumulated higher amounts of simazine in the flow-through system than in the static system. Large amounts of trifluralin were accumulated by all organisms in the flow-through system with most of it being the parent compound.

The uptake and elimination of propamocarb (a fungicide) by bluegill and channel catfish was investigated by Gray and Knowles.[118] The BCF for propamocarb was 1.5 and 3.0 for fillet and offal tissue, respectively, and the biological half-life was <7 d. The BCF for channel catfish exposed for 28 d to soil treated with the fungicide was 40 and 43 for fillet and offal tissue, respectively. As with the bluegills the half-life was <7 d. The uptake, distribution, and metabolism of thidiazuron in bluegills and channel catfish were investigated by Knowles *et al.*[119] Bluegills exposed to a continuous-flow concentration of 0.1 mg/l of ^{14}C-thidiazuron contained a maximum concentration of 5.4 μg/g in fillet tissue after one day of exposure. Tissue concentrations declined to about 1 μg/g after 28 d of exposure. When bluegills were injected intraperitoneally with ^{14}C-thidiazuron, metabolism and elimination were relatively rapid. A maximum level of 2.5 ng/g in offal tissue was detected after 7 d when channel catfish were exposed to 5.0 kg of soil containing 0.15 μg thidiazuron-^{14}C/g. In fillet tissue, radioactive material did not exceed 0.5 ng/g. Methemoglobin formation in suspensions of erythrocytes of white-spotted char (*Salvelinus leucomaenis*) induced by the nitroso-derivatives of biphenyl ether herbicides (2,4-dichloro p-nitrobiphenyl ether and 2,4,6-trichloro-p-nitrobiphenyl ether) and their analogs was investigated by Miyauchi *et al.*[120] All the nitroso-derivatives induced methemoglobin formation, and the activity of methemoglobin formation was the greatest in nitrosobenzene followed by p-nitrosobiphenyl ether, 4-chloro-p-nitrosobiphenyl ether, 2,4-dichloro-p-nitrosobiphenyl ether, and 2,-4,6-trichloro-p-nitrosobiphenyl ether. This order of potency was dependent on the number of chlorine atoms rather than on its position of substitution in the phenoxy group. The results from a similar study[121] indicated that char liver homogenates had the ability to metabolize p-aminobiphenyl ether to methemoglobin-forming metabolites although the activity was very low. The sensitivity of erythrocytes to the methemoglobin-forming metabolites was the lowest in char followed by the rat and was highest in human erythrocytes.

Piscicides and Anesthetics. Water samples taken 2 days after a mass fish kill of white amur (*Ctenopharyngodon idella*) in a pond in Orange County (Fla.) were analyzed by Martin and Martin.[122] Chloroform extracts were analyzed by HPLC and contained rotenone at a concentration of 1.8 mg/l. The origin of rotenone in the water was unknown. Orciari[123] used bioassays to determine the resistance to rotenone of golden shiner (*Notemigonus crysoleucas*) from Ball Pond (Conn.) which had six successive rotenone reclamations carried out from 1957 to 1974. The 24-h LC_{50} values of Noxfish are given

in Table 1. Based on LC_{50} values, golden shiners from Ball Pond were 4 to 7.1 times more resistant to rotenone than were golden shiners from seven control ponds. The authors concluded that fish from Ball Pond developed increased resistance to rotenone through the selective forces of reclamation. The effect of rotenone on the metabolic rates of rainbow trout using automated respirometry with increasing velocity steps was determined by Skadsen *et al.*[124] At a rotenone concentration of 350 μg/l (96-h LC_{50} value), fish showed reduced active metabolism and relative critical speed but no significant change in standard metabolism. At a concentration of 70 μg/l, standard metabolic rate was elevated but there was no significant change in standard metabolism or relative critical speed. Metabolic scope (active minus standard metabolic rate) was reduced at all concentrations. Gilderhus[125] conducted field trials in four lakes to evaluate the efficacy of Antimycin for control of larval sea lamprey (*Petromyzon marinus*). In the three lakes receiving a treatment of 150 g/ha (active ingredient), about 90% or more of the caged and wild lamprey were killed. The less than adequate control obtained with 75 g/ha in Ocqueoc Lake was assumed to be because of the low water temperature of 10°C. Some small fish, mostly bottom-dwelling, were killed by the treatments. The aquatic invertebrate community, fish populations, and brown trout population dynamics of a Wisconsin trout stream were studied by Avery[126] for 2 years before and 2 years after treatment with Antimycin A to eradicate a forage fish population consisting primarily of suckers, minnows, and darters. Significant improvements occurred in trout growth, standing crop, and production after removal of up to 1 445 kg/ha of forage fish. However, survival of brown trout did not improve following the treatment or after a reduction of the stocking density by 50%.

Niblett and McKeown[127] studied the comparative sensitivity of four age groups of raibow trout embryos to 24-h exposures of field grade (35.7% active) 3-trifluoromethyl-4-nitrophenol (TFM). Six hours, 10, 21, and 32 d after fertilization, embyros were exposed and returned to clean water for 46 to 50 d. LC_{50} values ranged from 4 mg/l for 6-h-old embryos to 43 mg/l for 10-d-old embryos (Table 1). Hinton and Eversole[128–130] conducted static toxicity tests with some commonly used chemicals in American eel (*Anguilla rostrata*) culture (Table 1). In general, younger glass eel stage (55 mm) was more sensitive than the black eel stage (97 mm) to 10 chemicals, and the larger yellow-phase eels (114–340 g) were the most resistant to four of five of the chemicals. Mitchell and Plumb[131] examined the acute toxicity of Furanace to channel catfish and determined the optimum treatment for channel catfish infected experimentally with *Aeromonas hydrophila*. The 96-h LC_{50} value was 0.94 mg/l, and the most effective treatment level of *Aeromonas hydrophila* was 2 mg/l for 6.5 h. A study to test the efficacy and toxicity of an iodophore used to disinfect muskellunge (*Esox masquinongy*) eggs incubated under hatchery conditions was described by Schachte.[132] The iodophore (Povidone-Iodine) was used for 10 min at concentrations of 100, 28, and 13 mg/l (active I_2) on water-hardened eggs. There were no significant effects on egg development to the eyed stage or to hatch among treatments or control. No toxic effects of iodophor were found at concentrations of active I_2 considered efficacious for salmonids. In an investigation of the disinfectant chloramine-T for control of bacterial gill disease, From[133] determined concentrations which were necessary to cure gill disease as well as concentrations that were toxic to rainbow trout. In non-renewal, static exposures ranging from 6 min to 12 h followed by observation for 1 week in flow-through aquaria without exposure to the toxicant resulted in maximum tolerated doses from 20 to 325 mg/l. The most effective concentration for control of bacterial gill disease was 4.5 to 6.5 mg/l.

Smit *et al.*[134] assessed the effects of MS-222 and neutralized MS-222 on the hematology of three freshwater fish. MS-222 produced the greatest hemoconcentration effects in the teleost (*Sarotherodon mossambicus*) and the least in trout. The use of neutralized MS-222 improved the hematological profiles and resulted in stabilization of acid-base parameters and RBC sizes and numbers. Trout were the most susceptible to the stress of MS-222 anesthesia. The effects of MS-222 on the ventilatory and heart rate response of rainbow trout exposed to environmental hypoxia were investigated by Daxboeck and Holeton.[135] Addition of MS-222 in concentrations up to 3.3 mg/l in normoxic water caused no significant changes in the mean resting heart or ventilation rates. At higher concentrations, there was an increase in mean resting heart rate but not in the ventilation rate. Concentrations ≥ 66.7 mg/l caused the ventilation rate to increase but not the resting heart rate. The extent of hypoxic response of decreasing heart rate and increasing ventilation rate was reduced with increased MS-222 concentrations.

Hill *et al.*[136] studied the combined effects of acute temperature changes and n-butanol on a conditioned response in goldfish acclimated to 20°C. The concentration of n-butanol required just to block the conditioned response was maximal (1 300 mg/l) at about 18°C and either raising or lowering the temperature reduced the required concentration. At 20°C, the threshold values for ethanol, butanol, and hexanol were 4 600, 780, 72 mg/l, respectively. The concentrations for zero success were 12 000, 1 300, and 130 mg/l, respectively. The effect of a single treatment of 4.4 mg/l of tetracycline hydrochloride on the critical thermal maximum of common shiner (*Notropis cornutus*) was measured by Schubauer *et al.*[137] Fish exposed to tetracycline hydrochloride for 3 weeks and acclimated at 15°C had a critical thermal maximum of 32.0°C that was not significantly different from that of 31.9°C for fish acclimated in the control tank.

Inorganics. Mercury content of walleye (*Stizostedion vitreum vitreum*), northern pike, and lake whitefish (*Coregonus clupeaformis*) from Ball Lake (north-western Ontario) was measured before and after Hg discharge controls for a chlor-alkali plant were established.[138] Substantial reductions in Hg residues were found in the fish after discharges were controlled. It was suggested that Hg in suspension in the water column may be a more important consideration for Hg uptake by fish than sediment Hg concentrations. Mercury concentration in the muscle of rainbow trout stocked in Antelope Reservoir (Oreg.) increased linearly for 5 months.[139] After 8 months it leveled off at a concentration similar to that of indigenous fish. Fish in a new reservoir (Cedar Lake) were found to have high Hg concentrations, some exceeding the FDA standard. Cox *et al.*[140] concluded these were from natural amounts in the soil and not from influent water. Mercury concentrations were higher in larger fish and particularly in those higher in the food chain. Wissmath and Kreuzer[141] measured Hg concentrations in field-collected fishes, correlated the length and

weight of sample fish with the Hg-to-size ratio, and found that the Hg contents of specimen groups were significantly higher in liver than in muscle tissue. Mercury concentrations in trout (*Salmo trutta* L.) 3 000 m below a Hg discharge were approximately 3 times higher than levels found in trout above the outlet.[142] The Hg content in eels (*Anguilla anguilla* L.) was not significantly different above and below the discharge. Mercury concentrations were measured in fish from the southwestern Lake Erie region, that were principal food sources for herons and egrets.[143] It was concluded that goldfish, freshwater drums (*Aplodinotus grunniens*), perch, bowfin (*Amia calva*), and alewife (*Alosa pseudoharengus*) were the most important contributors of Hg to herons and egrets. Moore and Sutherland[144] determined the total Hg in sediments, water, and fish from Giauque Lake and Thompson Lake in northern Canada during 1977 and 1978. Both lakes had formerly received Hg-laden discharges. Water concentrations of Hg in both lakes were near the detection limit but bottom sediments contained as much as 1 300 mg/kg. Lake trout had the highest muscle residues (3.79 mg/kg) in Giauque Lake whereas northern pike contained 1.69 mg/kg in Thompson Lake. It was concluded that northern pike were still accumulating Hg from tailings deposited in Thompson Lake 30 y previously and that contamination in only a small part of the lake may have resulted in high levels in fish throughout the lake. The levels of methylmercury generated in sediments and its partitioning to various compartments in aquatic ecosystems, including uptake by goldfish, were observed by Akagi *et al.*[145] The difference in distribution between fish-containing systems and fish-free controls was not significant, indicating that the rate-limiting step for methylmercury accumulation was its uptake by fish from the surrounding water. Mercury levels in Labrador freshwater and anadromous fish species collected in 1977 and 1978 were examined by Bruce *et al.*[146] Approximately 6 000 tissue samples representing 14 fish species were analyzed. Mean Hg values were highest in lake trout and nothern pike and exceeded 0.5 μg/g. Fish species such as whitefish, suckers (*Catostomus* sp.), and brook trout contained residues generally <0.5 μg/g. Mercury content in whole freshwater and saltwater fish in Guatemala was analysed by Ramos *et al.*[147] Residues of Hg in freshwater shellfish and finfish ranged from 0 to 0.31 μg/g. It was concluded that Hg contamination in Guatemalan fish did not constitute a large problem. A survey of Hg content of 19 fish species from 18 locations in the coastal and freshwaters of Papua (New Guinea) showed that the Hg content was generally well below the 0.5 μg/g limit recommended by the World Health Organization, with the exception of the barramundi (*Lates calcarifer*) caught in the Fly River system. The mean value for this species was 0.52 μg/g.[148] A positive correlation between Hg content and fish weight was found. Mercury, methylmercury, and Se were measured in field-collected pike, perch, and pike-perch (*Stizostedion lucioperca*).[149] Fillet samples of these fishes often contained Hg at concentrations above 1.0 mg/kg, although mean residue concentrations were less than 1.0 mg/kg. About 90% of the Hg in edible portions was present as methylmercury. Perch liver tissues showed a significant correlation between methylmercury concentration and Se concentration. Levels of Hg and Se in fish, sediments, and water of Lake Dufault and Lake Duparquet in northwestern Quebec were determined by Speyer.[150] Samples of muscle tissue of northern pike from Lake Dufault had low Hg concentrations (0.01 to 0.03 mg/kg) and higher Se concentrations (1.1 to 3.0 mg/kg), whereas fish in Lake Duparquet had elevated Hg levels (0.17 to 1.38 mg/kg) but lower Se levels (<0.20 to 0.62 mg/kg). Other analyses indicated elevated concentrations of both Hg and Se in the sediments but only detectable levels in water.

Selenium concentrations of 50 to 70 μg/g were found in rainbow trout and brown trout collected from natural waters containing high Se concentrations (12 to 13 μg/g).[151] Effects of transfer-RNA methylation in fish chronically exposed to Se were examined, and direct nucleoside analyses of mature tRNAs isolated from trout livers having both low and high Se concentrations showed no evidence of methylation aberrations. A reduction in fish population in a North Carolina lake was attributed to elevated Se concentrations, the source of which was return of fly-ash sluice water from a coal-fired power plant.[152] Tissue concentrations of Se were elevated in fish that accumulated Se via the food chain to which Se was introduced by plankton-Se uptake. The use of electronically measured respiration of fish exposed to various toxicants present in the Rhine River was reported by Sloof.[153] For 13 materials rainbow trout had sensitivities from 0.025 mg Cd/l to 20 mg chloroform/l in comparison to zebrafish (*Brachydanio rerio*) that had sensitivities of 2.5 mg Cd/l and 100 mg chloroform/l. Hegi and Geiger[154] measured Hg, Cd, Cu, Pb, and Zn concentrations in fish collected from two Swiss lakes. Mercury content was found to be higher in muscle tissue than in livers whereas the reverse was true for the other metals. Concentrations of Cd, Pb, Cu, Mn, and Zn were measured in stocked bluegill and eight resident fish species in a shallow pond in South Carolina.[155] It was suggested that concentrations of Cu, Mn, and Zn were homeostatically controlled in liver tissue, axial musculature, and whole body. Concentrations of all metals in axial muscle tissue and whole body of stocked bluegill were fairly constant over a 200-d period. Fish collected from inland waters in West Germany were analyzed for Pb, Cd, As, Zn, and Cu.[156] Metal concentrations in the liver were higher than in muscle and concentrations decreased with increasing distance from a metal emission source. Perch had the highest metal concentrations and pike the lowest; roach, chub, and tench were intermediate. Brook trout above and below the wastewater outfall of Göttingen (Ger.) were analyzed for Cd, Cu, Hg, Ni, Pb, and Zn.[157] Statistically significant increases of some of the metals were found below the city: Cd in flesh; Cd, Hg, and Zn in liver; and Cd, Cu, and Zn in total fish. Gächter and Geiger[158] examined the fate of Cd, Hg, Cu, Zn, and Pb in the food chain containing phytoplankton, zooplankton, chironomids, and fish fry in an unpolluted and an artificially metal-polluted freshwater system. On a dry-weight basis, metal concentrations were highest in phytoplankton and periphyton samples, lower in zooplankton, chironomidae, *Sialis*, and lowest in bream fry and eggs and larvae of the European lake trout (*Trutta fario* f. *lacustris*). Results suggested that inorganic metals do not bioaccumulate up a food chain. Vinikour *et al.*[159] measured whole body concentrations of Zn, Cu, Cd, and Pb in selected fish species from a relatively unpolluted section of the Fox River in Illinois and found that whole body concentrations on a μg/g basis did not generally increase with fish size. Tissues from field-collected carp, largemouth bass, and bluegill were analyzed for Cd, Cr, Cu, Pb, Zn, and total Hg.[160] It was found that Hg accumulated in

fillet tissue as a function of age, weight, and length. Concentrations of Cu and Zn in liver tissue were typically higher than those in fillet tissue. Concentrations of Cd and Zn were measured in yellow perch collected from an Indian lake to determine effects of these metals on fish growth.[161] Cd concentrations were inversely correlated with growth, whereas Zn concentrations were not. RNA-DNA ratios were found to be sensitive indicators of fish growth. In a related study,[162] samples of fish from an industrially contaminated part of a lake and an uncontaminated area revealed that fish from the uncontaminated area had higher weight/length ratios. Body burdens of Cd increased in fish as condition and RNA-DNA ratios decreased but no relationship was found with Zn. Adams *et al.*[163] determined the levels of Cd and Zn in water, sediment, plants, fish, and clams from Trimble Creek receiving water from the industrial-contaminated Palestine Lake in Indiana. The mean levels of Cd and Zn in species of fishes from Trimble Creek were 6.02 and 145 μg/g, respectively, slightly lower than those found in the west basin of Palestine Lake. However, it was concluded that this stream was noticeably contaminated with Cd and Zn, this being attributable to the contamination from the lake. Furr *et al.*[164] analyzed aquatic organisms including bullhead (*Ictalurus nebulosus*) from a farm pond in Lansing (N. Y.) contaminated with coal fly ash. Values in bullhead liver ranged from 0.1 μg/g of Yb, Sm, and Lu to 13 970 μg/g of K. Selenium in bullheads ranged from 1.7 to 5.2 μg/g in the flesh and 8.2 to 9.2 μg/g in the liver. Arsenic concentrations were measured in six Lake Michigan fish species.[165] Arsenic concentrations in tissues of lake trout correlated with number of years in the lake, and in both lake trout and smelt (*Osmerus mordax*) correlated with length. No correlation with age or length was found for alewife or yellow perch. The effect of Al speciation on brook trout and white sucker (*Catostomus commersoni*) in dilute acidified waters was studied by Driscoll *et al.*[166] to characterize Al chemistry in acidified waters of the Adirondack region of New York State and to assess the relative toxicity of soluble Al species to fish. Additions of fluoride or citrate decreased the toxicity of Al solutions to brook trout with organic complexation having a greater effect on Al toxicity than fluoride complexation. Mortality rates of white sucker fry in three naturally acidified Adirondack waters (pH 5.0) appeared to be determined by the levels of labile inorganic Al present. Elwood *et al.*[167] studied bluegill, largemouth bass, goldfish, and mosquitofish for Cr uptake from a lake contaminated with chromates. There was a negative correlation with fish size and chromium concentration in muscle.

The acute toxicity (24- and 48-h) of As to a tropical freshwater fish (*Puntius sophore*) was determined.[168] It was suggested that mortality was a result of respiratory and circulatory failure. In a partial life cycle test (4.5 months) with Cd and Atlantic salmon, Grande and Andersen[169] found that there was complete mortality at >5 μg Cd/l whereas there was no significant mortality at 1 μg Cd/l (Table 1). The influences of hardness constituents on the acute toxicity of Cd to brook trout were studied by Carroll *et al.*[170] Of the three major constituents characteristic of hard water (Ca^{2+}, Mg^{2+}, and CO_2) Ca^{2+}, introduced either as sulfate or carbonate, was the major source of protection against Cd toxicity and this protective effect was observed in the absence of significant Cd precipitation. Calamari *et al.*[171] studied the influence of hardness on the acute toxicity of Cd to rainbow trout fry. Acute toxicity was found to increase with a reduction in water hardness. Results showed that similar concentrations of the same form of cadmium (Cd^{2+}) gave different levels of mortality and that similar acute toxic effects were caused by different amounts of ionic form (Cd^{2+}). Fish acclimated in hard water (320 mg/l as $CaCO_3$) but tested in soft water (20 mg/l as $CaCO_3$) reacted in an intermediate way. The relationship between chloride cells and water hardness with respect to fish tolerance was discussed. The mortality of *Heteropneustes fossilis* was tested at 250 mg $CdCl_2$/l at various temperatures from 22 to 36°C.[172] Mortality increased with increased temperatures. An increase in temperature was found to increase the toxicity of Cd to rainbow trout.[173] Fish acclimated to 6°C survived in lethal concentrations of Cd approximately twice as long as fish acclimated to 18°C. Plasma calcium concentrations declined steadily in fish exposed to 0.3 mg Cd/l, and this was postulated to be the direct cause of Cd-induced mortality. Adverse effects of $CdCl_2$ and NaBr (Table 1) on the guppy, *Oryzias latipes*, and *Xenopus laevis* were reported by Canton and Slooff.[174] EC_{50} concentrations were 180 mg NaBr/l (reproduction) and 225 mg NaBr/l (mortality and abnormal behavior) for the guppy and 50 μg Cd/l (mortality) and 20 μg Cd/l (mortality and abnormal behavior) for *Oryzias latipes*. For *Xenopus laevis*, EC_{50} values were 1 500 μg Cd/l (mortality) and 650 μg Cd/l (larval development). Fathead minnow survival was affected at a Cr(VI) concentration of 3.9 mg/l in chronic studies conducted by Pickering.[175] The lowest Cr concentration (0.018 mg/l) affected growth, whereas reproduction and hatchability of eggs were not affected at any concentration tested. The effects of hardness, alkalinity, and pH on the toxicity of Cu to rainbow trout were examined by Miller and Mackay.[176] In low water hardness (12 mg/l), the incipient lethal concentration (ILC) of Cu was not affected by a 5-fold change in medium alkalinity (10 to 50 mg/l) but increased the ILC by 1.8 times in harder water (98 mg/l). The ILC increased with increased hardness regardless of alkalinity. At pH less than 5.4, the toxicity of Cu and hydrogen ion appeared to be antagonistic whereas there appeared to be synergism between the two above pH of 5.4. Horning and Neiheisel[177] reported that Cu adversely affected fry survival, growth, and reproduction in a chronic toxicity test with the bluntnose minnow (*Pimephales notatus*). The authors reported a maximum acceptable toxicant concentration (MATC) between 4.3 and 18.0 μg Cu/l. The relationship between survival, overturning, and lethal exposure time for fish exposed to Cu in acute tests was studied by Tsai.[178] All three endpoints decreased as exposure time increased at the concentrations tested (1 to 279 mg/l). The median lethal exposure time was shorter than the median overturning time and much shorter than the median survival time. The overturning period for fish overturned and killed in Cu solutions was independent of exposure time, survival time, and Cu concentration. Shaw[179] compared three responses, median survival, 10% mortality, and 10% turnover time, of rainbow trout exposed to Cu and CN as monitoring tools for detecting pollution. For all concentrations of the chemicals (0.5, 1.0, and 2.0 g Cu/m^3 and 0.3, 0.5, 1.0, and 5.0 g CN/m^3), the times for turnover were the least. It was indicated that this method may be generally applicable for detection of gross pollution by hazardous chemicals. Acute toxicity of Cu (as $CuSO_4 \cdot 5H_2O$) and Hg (as

$HgCl_2$) to guppy and *Labeo rohita* was studied in relation to fish size.[180] Fry were found to be more susceptible than fingerlings and female guppies were more resistant than males of the same size. Andros and Garton[181] reported that acute exposures of northern squawfish, *Ptychocheilus oregonensis*, to Cu, Cd, and Zn resulted in 96-h LC_{50} values (Table 1). This showed northern squawfish that compete for food with salmonids as well as prey upon them were more tolerant than chinook salmon (*Oncorhynchus tshawytscha*) and steelhead (*Salmo gairdneri*) to Zn and Cd, but were equally sensitive to Cu. Chiselmouth, *Acrocheilus alutaceus*, were more resistant than northern squawfish to Cu. Acute static tests were conducted by Pant *et al.*[182] to compare the toxicity of Cu and Zn to *Puntius conchonius*. Copper was appreciably more toxic than Zn (Table 1). Thompson *et al.*[183] determined the 96-h LC_{50} values for Cu and Zn individually and in combination to bluegill sunfish (Table 1). Copper and Zn mixed at a 1:1 toxicity ratio exhibited additive effects. The effects of mixtures of Cu and Zn to eggs, alevins, and swim-up fry of chinook salmon was investigated by Finlayson and Verrue[184] using 96-h and 83-d tests. Estimated "safe" levels of Cu and Zn based on growth from eggs to fry were 11 μg Cu/l and 83 μg Zn/l and additive effects resulted when these metals were mixed. Acute toxicity of $CuSO_4$, $ZnSO_4$, and $FeCl_3$ were determined for the guppy.[185] The 48-h LC_{50} values were 3 times lower for the metals in combination than for each metal alone (Table 1). Zebrafish eggs were incubated for 24 h in solutions of copper nitrate (58 μg Cu/l), lead nitrate (58 μg Pb/l), and a mixture of Cu and lead nitrates (58 μg Pb/l + 8 μg Cu/l).[186] Some of the eggs were then dechorionated and switched from Cu to Pb solutions and vice versa. Embryos incubated first in Cu then dechorionated before transfer to Pb survived longer than those from Pb to Cu. Embryos dechorionated after 24-h Cu exposure and then exposed to Pb were abnormal. Those reared in Pb, dechorionated, then transferred to Cu did not survive and embryos exposed to a combination of Cu and Pb did not hatch. Photoperiod acclimation and circadian variations in tolerance of juvenile rainbow trout to Zn were examined by McLeay and Munro.[187] The results showed that for both conditions of photoperiodic acclimation, the within-replicate variance was less than the hour-by-hour variance for time-to-death values, indicating that circadian variations in tolerance to Zn were evident. Other results suggested that the time to death for fish acclimated to a given photoperiod was consistent if the time of day that the exposure was initiated was the same.

Mahajan and Juneja[188] studied the combined effect of Hg in the diet and the water on *Channa punctatus*. Mercury (34 μg/l) in the water caused greater fish mortality than in the diet (0.1% Hg). The greatest mortality effect, however, occurred when fish were exposed to Hg in diet and water simultaneously. Fish exposed to combined sources of Hg also showed the greatest loss in weight after the 45-d exposure. The acute toxicity of $HgCl_2$ and pentachlorophenol to *Channa gachua* was determined by Hanumante and Kulkarni.[189] The 96-h LC_{50} values are shown in Table 1. Pentachlorophenol was found to be more toxic than $HgCl_2$ after 96 h. The effect of selenium dioxide pretreatment on the acute toxicity of $HgCl_2$ to embryos of Japanese ricefish (*Oryzias latipes*) was studied by Bowers *et al.*[190]. Selenium pretreatment did not decrease the toxicity of $HgCl_2$ alone (Table 1). In 96-h exposures of Se as sodium selenite (Na_2SeO_3) and sodium selenate (Na_2SeO_4), susceptibility of three warmwater organisms was in the order *Hyallela azteca* (most susceptible) > *Daphnia magna* > fathead minnow.[191] In chronic tests, daphnid survival and reproduction were unaffected at concentrations as high as 0.28 mg Se/l. Concentrations up to 40 mg Se/l had no effect on the hatchability of fathead minnow eggs but concentrations above 15 mg Se/l significantly reduced egg incubation times. The effect of Se (selenite) on carp was examined by Sato *et al.*[192] Residues in whole body tissue increased with increased exposure concentration but decreased when fish were returned to clean water. The biological half-life of Se was about 28 days and selenium concentrations in the kidney, liver, gall bladder, and gill were higher than those in the heart, bone, and muscle. The acute toxicity of Aroclor 1254 and As_2O_3 to ciscoes (*Coregonus* sp.) were determined (Table 1).[193] Combinations of the two toxicants were more toxic than either alone. Concentrations of PCBs, As, and DDT plus DDE in cisco tissues were reported to be 1.2 to 2.3, 0.75 to 0.84, and 0.67 to 1.4 μg/g, respectively. Spehar *et al.*[194] tested toxicity and bioaccumulation of As(III), As(V), sodium dimethyl arsenate, and disodium methyl arsenate at concentrations of 100 and 1 000 μg As/l to stoneflies, snails, amphipods, daphnids, and rainbow trout. Arsenic(III) at a concentration of 1 000 μg/l caused significant mortality to amphipods but other animals were not affected by this or other As compounds. Body residues of As were found at higher concentrations than the controls in stoneflies, snails, and daphnids but not in amphipods and trout. A study on the metabolism of inorganic ^{74}As to organoarsenicals in rainbow trout was conducted by Oladimeji *et al.*[195] Techniques applied to muscle, liver, kidney, blood, bile, and urine in fish given an oral dose of 20 μCi ^{74}As/500 mg diet showed that conversion to an organic form with the same R_f as ethylarsenobetaine was rapid and extensive. The ratio of total organic ^{74}As to inorganic ^{74}As increased with time in all tissues, with the organic fraction accounting for about 50% at 6 h and over 80% of 24 h.

Cadmium uptake by fingerling perch at two temperatures increased with time and resulted in doubling of the uptake rate with an increase in temperature of 10°C.[196] Although no equilibrium in the uptake of Cd was reached after 6 weeks, elimination of Cd from fish transferred to natural lake water appeared rapid. Analysis of the tissues showed that the liver and kidney contained the highest Cd residues. Blickens[197] subjected brown bullheads to 61.3 mg Cd/l (as $CdCl_2$) for either 2 or 4 h followed by varying periods of up to 2 months in Cd-free water. Cadmium residues in gills were measured and it was found that the fish did not clear accumulated Cd totally even after 2 months in Cd-free water. Histopathological changes observed in gills after exposure included hemorrhage, desquamation, edema, stasis, and inflammation. The effects of acclimation temperature (7 to 29°C) and DO concentration (4.5 to 8.5 mg/l) were tested in relation to accumulation of Cd, Cr, and Pb in bluegill sunfish.[198] Accumulation of Cr and Pb in gill tissue appeared to be related to DO level whereas Cd, Cr, and Pb accumulation in other tissues were temperature-related. Correlation patterns indicated that Cd and Pb were more metabolically active than Cr. Rainbow trout, fathead minnows, and brown shrimp (*Penaeus*) were exposed to 0.01, 1.0, and 10 mg/l concentrations of Pb, Hg, Cu, or Zn for 96 h to determine whether metal residue levels in exposed

animals correlated with acute lethality.[199] It was found that heavy metal accumulations in whole body tissue were not directly correlated with acute lethality and no useful threshold of accumulation existed. Metal exposure concentrations in the water were more important determinants of both accumulation and toxicity. The uptake of ^{60}Co by carp via food and water was part of a series of studies on uptake of ^{60}Co by organisms from different trophic levels.[200-202] Cobalt content of edible portions of carp was low, with highest concentrations found in the kidney. Cobalt residues were rapidly eliminated, most quickly from the digestive tract and most slowly from the gills and liver. Cobalt contamination from water or from both food and water resulted in the same ^{60}Co tissue concentrations. Body size relationships for accumulation and tolerance of fish exposed to Cu were examined by Anderson and Spear.[203] The LC_{50} for pumpkinseed sunfish (*Lepomis gibbosus*) increased disproportionately with body weight and varied as the reciprocal of Cu accumulation rate in gills. However, LC_{50} and accumulation rate for rainbow trout did not change with body weight. Manganese uptake in bluegill was not significantly affected by DO.[204] A water temperature of 10°C decreased uptake although uptake at 20 and 30°C was similar. Uptake of Mn from prey fish by largemouth bass was greater than from a water route. It was suggested that the uptake from food and water in wild fish would each contribute large fractions to bass. Hatchery northern pike were fed field-collected carp with high Hg concentrations in tissue (1.10 to 0.14 μg Hg/g).[205] Pike retained an average of 19% of methylmercury ingested from carp. The total Hg content in fish-fed mixtures with a sulfite yeast supplement was determined by Svobodová *et al.*[206] A limiting value of 0.1 mg Hg/kg was slightly exceeded in the muscle of 2-year-old carp reared on a mixture containing 15% of sulfite yeasts. Phillips *et al.*[207] determined that Hg concentrations in fish flesh increased with age of the fish. All older fish exceeded the 0.5 μg Hg/g guideline for safety. Fish studied included northern pike, walleye, sauger (*Stizostedion canadense*), black crappie, and white crappie (*Pomoxis annularis*). Mercury methylation in the intestinal tract of several species of freshwater fish was reported by Rudd *et al.*[208] These investigators used a new radiochemical method and suggested that the conversion of inorganic to organic Hg in the intestine was probably brought about by bacterial action. Stary and Kratzer[209] derived equations for calculating concentrations of methylmercury, phenylmercury, and inorganic Hg in *Poecilia reticulata* using experimentally determined cumulation and release constants. The authors concluded that uptake and depuration could be used for calculation of concentration of individual Hg species in fish under different conditions. The bioaccumulation and bioamplification of Hg compounds in *Gambusia affinis* were studied by Boudou *et al.*[210] Methylated Hg was more toxic and more easily bioaccumulated than inorganic Hg. Both measurements were increased with increased water temperature. Bioamplification was illustrated in a trophic food chain test with algae, *Daphnia*, and fish. In a related study, Boudou *et al.*[211] investigated the bioaccumulation and amplification of Hg compounds in a third-level consumer (*Salmo gairdneri*) in addition to other organisms in the food chain and reported preponderance of trophic contamination, as compared with direct contamination from the water.

An increase in water hardness from 40 to 440 mg as $CaCO_3$/l increased the tolerance of channel catfish to ammonia (Table 1).[212] Hematocrit, percent plasma protein, plasma chloride, tissue chloride, and blood pH were not affected by 12-h exposures to 1.025 mg un-ionized NH_3-N/l. Plasma sodium concentrations were similar in exposed fish compared to controls at pH 7, but lower at pH 8. An investigation of gill sialic acid content as an index of environmental stress in rainbow trout showed that a significant increase in sialic acid concentration occurred in fish exposed to ammonia.[213] However, this increase was influenced by the concurrent increase in pH, as indicated by a gill sialic acid concentration higher than the controls, but slightly lower than at the same pH with ammonia, when sodium hydroxide was used. Osmoregulatory effects of acute Cd toxicity to goldfish were studied by Busacker.[214] Acute exposure to Cd reduced blood sodium and chloride, and elevated blood potassium, cortisol, and hematocrit. Following a single subcutaneous injection of ^{109}Cd, tissue Cd concentrations were highest in kidney, pronephros, liver, and spleen. The biological half-life ranged from 46 h to >5 y. Calcium and inorganic-P excretion rates increased in carp orally dosed with Cd.[215] Overexcretion of Ca was attributed to degeneration of the renal tubules. Susceptibility to Cd toxicity by the medaka (*Oryzias latipes*) was correlated with the hepatosomatic index and not sexual maturation as induced by photoperiod.[216] Srivastava and Mishra[217] described pathological changes in blood of *Colisa fasciatus* following acute exposure to 100 mg Cd/l. After 90 h of exposure, lymphocytosis, and anemia was induced, with simultaneous regeneration of red blood cells. The treatment also evoked thrombocytosis with concomitant hypercoagulability of whole blood. The 96-h LC_{50} value for this species was 126 mg/l. Beattie and Pascoe[218] intraperitoneally injected rainbow trout with 0.5 ml of a 300 mg Cd/l saline solution on four successive days (total dose = 3.0 mg Cd/kg body weight). From examination of liver tissue extracts from exposed fish, it was suggested that treatment with Cd induced synthesis of a protein, similar to metallothionein, that was able to bind with and effectively detoxify Cd. Male adult *Pimelodus maculatus* were intraperitoneally injected with a solution of $CdCl_2$ (hydrated) in a single dose of 10 mg/kg and sacrificed after 3 d.[219] Electron microscope examination of liver sections of exposed animals showed hypertrophied Golgi complexes, suggesting an increase in the synthesis of proteins from the rough endoplasmic reticulum. Lysosomal activity in the liver of this fish increased after a 24-h exposure to Cd and was related to shape, distribution, and different functional degrees of organelle condensation. No effect was observed on survival, growth, and histology of rainbow trout exposed to Cd compounds via the water at a concentration of 4 μg Cd/l for 10 weeks or to fish exposed to Cd compounds via food at a concentration of 10 mg Cd/kg for 12 weeks.[221] However, fish orally exposed to 100 mg cadmium stearate/l showed some histological changes. Total Cd loads decreased in fish orally exposed after discontinuation of exposure whereas those in fish exposed through the water virtually remained unchanged during this time. Translocation of Cd in tissues showed different patterns of absorption after withdrawal from exposure. The effect of a sublethal Cr freshwater exposure to juvenile coho salmon on salinity tolerance and serum osmolality was investigated by Sugatt.[222] After transferring Cr-exposed fish to salt water survival decreased and serum osmolality was significantly higher than in control fish. Juvenile coho salmon were

exposed to sodium dichromate (0.5 mg Cr/l) for two weeks then injected with a virulent strain of *Vibrio anguillarum*.[223] Disease resistance and agglutinin production of coho salmon against *V. anguillarum* were impaired by sublethal Cr exposure. Adult *Colisa fasciatus* females were exposed for 90 h to 35 mg Cr/l (as chromium trioxide) and hematological characteristics induced by the Cr exposure were examined.[224] Effects of the acutely sublethal exposure were pronounced increases of circulating erythrocytes, hematocrit, clotting time, and hepatosomatic index compared to controls. Leukopenia, thrombocytopenia, and increased erythrocyte sedimentation rate was also observed. There was no change in hemoglobin nor in the number of circulating immature erythrocytes or large lymphocytes. Adult female *Colisa fasciatus* were exposed to 3 mg Cu/l (as copper nitrate) for 90 h.[225] Hematological studies showed Cu-induced increases in total red blood cell count, hematocrit, hemoglobin, and number of erythrocytes and thrombocytes per 1 000 blood cells. The differential counts of small and large lymphocytes decreased slightly in exposed fish compared to controls, and clotting time and erythrocyte sedimentation rate decreased significantly. Total leucocyte and thrombocyte counts and the hepatosomatic index were not significantly altered by Cu exposure. Changes in hematocrit of rainbow trout exposed to various combinations of water hardness, pH, and Cu were observed by Waiwood.[226] In the absence of Cu, water hardness and pH did not significantly alter the hematocrit of fatigued fish. However, hematocrit increased with increased Cu concentration at pH 6.0, but at pH 7.9 the response to Cu was evident only at a hardness of 30 mg/l. Data on red blood cell counts and carcass and muscle moisture suggested that hematoconcentration was accompanied by a shift in water from blood to muscle.

Studies on intraperitoneal toxicity of Pb to zebra cichlid (*Cichlasoma nigrofasciatum*) development were conducted by Ozoh.[227] Concentrations of 50 to 600 mg/l were used and injected at 1 ml/100 g body weight. Responses to Pb were ovulation stress, loss of muscular coordination and balance, deformity, embryo mortality, and fry mortality and abnormality. Hematological effects and activity of the enzyme delta-aminolevulinic acid dehydratase (ALA-D) were studied in rainbow trout exposed to lead nitrate (10, 75, and 300 μg Pb^{2+}/l) for 30 d followed by 49 d of recovery in Pb-free water.[228] ALA-D activity was strongly depressed in erythrocytes, spleen, and renal tissue of fish exposed to all Pb concentrations. Fish exposed to 300 μg Pb^{2+}/l also exhibited anemia and basophilic stippling of erythrocytes. These effects persisted even after the recovery period; however no effects of Pb exposure on the white blood cells were found. The freshwater catfish, *Heteropneustes fossilis*, was subjected to a 3-h exposure to 50, 100, and 250 mg Pb/100 ml.[229] Lead-induced depletion in glycogen content in liver, kidney, and brain tissue occurred. In muscle tissues there was an initial depletion in glycogen at 50 mg Pb/100 ml and an increase at 100 mg Pb/100 ml, with muscle glycogen being lowest at 250 mg Pb/100 ml. The effect of exposure of *Channa punctatus* to $Pb(NO_3)_2$ (LC_{50} concentration of 6.8 mg/l and a sublethal concentration of 2.8 mg/l) was investigated by Sastry and Gupta.[230] An inhibition of activity of amino tripeptidase, glycylglycine, and leucyl-1-glycine was observed after 96 h and 15 and 30 d, whereas lipase activity was not affected. Results indicated that Pb-induced toxicity was more severe on a chronic basis than after acute exposure. Enzymes in the liver, stomach, and intestine of *Heteropneustes fossilis* were altered after a 30-d exposure to 2.8 mg $Pb(NO_3)_2$/l.[231] All enzymes studied except acid phosphatase were significantly inhibited in the liver and most enzymes were inhibited in the intestine, with enzymes in the stomach generally unaffected. The morphological and physiological effects of waterborne Pb and dietary-deficient and supplemented ascorbic acid on rainbow trout were reported by Hodson *et al.*[232] The authors found that, although aspects of Pb toxicity resembled ascorbic acid deficiency, there was no metabolic interaction between these factors. Eggs of zebrafish were exposed to 50 μg Pb/l for 24 h, dechorionated, and exposed to Pb for an additional 24 h.[233] A second group of eggs was exposed to 72 μg Pb/l for 24 h then transferred to Pb-free water. Irreversible damage to pigment pattern formation was caused by Pb exposure in the dechorionated embryos. Embryos subjected to 72 μg Pb/l did not exhibit significant changes in pigment patterns. The shell membranes appeared to provide some protection to embryos agains Pb. Catfish were exposed to 0.001% lead acetate for periods of up to 30 d.[234] Examination of hematological parameters of exposed fish showed the establishment of a microcytic hypocromic anemia. Ultrastructural investigations showed that the initial effects of Pb toxicity were fragmented and reduced cristae of erythroblast mitochondria. The development of black tails (an early indication of spinal deformity) occurred in rainbow trout at 22 μg Pb^{++}/l but not at 11 μg Pb^{++}/l.[235] Exposures lasted from the egg stage through 30 weeks post-hatch and levels were 5 times lower than reported levels for older fish. The effect of Pb and Zn and their mixture on the erythropoiesis of catfish was investigated by Garavini *et al.*[236] On the basis of hematological parameters, Pb-treated animals showed microcytic hypocromic anemia whereas Zn-treated fish showed a microcytic anemia. Mixture studies showed a protective action of Zn on Pb toxicity. Haider[237] found necrosis of sensory and supporting cells in the lateral line canal organs of rainbow trout following exposures to sublethal concentrations of Pb and Zn. No differences were found between the effects of Pb and Zn.

A comparative study of the effect of acute and chronic $HgCl_2$ treatment on the activity of some digestive enzymes of *Channa punctatus* was made by Sastry *et al.*[238] After 96 h of exposure to 1.8 mg/l, a slight elevation in maltase activity was recorded in all portions of the digestive system. Lipase and leucyl-1-glycine dipeptidase activity was inhibited. Chronic treatment of 0.30 mg $HgCl_2$/l significantly inhibited the activity of all the enzymes tested. In a similar study changes in the activities of some digestive enzymes of *Channa punctatus* exposed to 0.3 mg $HgCl_2$/l for 15 to 30 d were observed.[239] Glucose-6-phosphatase and lactase activity was significantly inhibited in the intestine and pyloric caeca after 15 d whereas maltase activity was not. Activities of three peptidases along with lipase were decreased in the parts of the digestive system examined. Results indicated that decreases in some enzyme activity were greater after 30 d of treatment than after 15 d. Sharma and Davis[240] studied the effect of CH_3HgCl on protein synthesis in the liver of carp. A concentration of 50 μg/l caused a 900-fold Hg concentration in liver and decreased protein synthesis as measured by rate of *in vivo* leucine incorporation after a 16-d exposure period. The concentration of free sulfhydryl groups was not significantly altered. Effects of an acute

lethal (1.8 mg/l) and a sublethal (0.3mg/l) concentration of $HgCl_2$ on the activity of some enzymes in the brain of a freshwater fish (*Ophiocephalus Channa punctatus*) were examined by Sastry and Sharma.[241] Activities of acid phosphatase and Na^+, K^+ adenosine triphosphatase were elevated in acutely exposed fish (4 d) but inhibited in longer exposures (15 to 30 d). The activities of glucose-6-phosphatase, succinic dehydrogenase, lactic dehydrogenase, pyruvic dehydrogenase, and cholinesterase were inhibited by all treatments. In another experiment,[242] *in vitro* effects of $HgCl_2$ and EDTA on activity of three phosphatase enzymes in the brain of *Channa punctatus* were observed. Maximum inhibition was observed in activity of glucose-6-phosphatase and was directly proportional to the concentration of $HgCl_2$ present. EDTA was capable of restoring the activity of all three enzymes. Restoration was gradual and its extent increased with increased EDTA concentration and time. An air-breathing fish, *Heteropneustes fossilis*, was more resistant towards Hg in a 2-wk exposure than was a non-air-breathing fish, *Sarotherodon mossambica*.[243] Effects of Hg toxicity on lysosomal enzymes (acid phosphatase, alkaline phosphatase, β-D-glucoronidase) and microsomal glucose-6-phosphatase in various tissues of *H. fossilis* and *S. mossambica* were different in the two fishes, but a definite stress condition was found in both leading to a significant impairment of metabolism. A solution of $HgCl_2$ was injected into another air-breathing fish (*Clarias batrachus*).[244] Analysis for alkaline phosphatase and acid phosphatase in the dendritic organ showed enhanced activity of the former and inhibition of the latter in experimental fish when compared with controls. It was concluded that Hg, although acting as a vascular poison, caused damage to accessory respiratory organs that proved fatal to fish. Injections of methylmercury, Cu, or methylmercury plus Cu to blue gourami (*Trichogaster trichopterus*) appeared to reduce the proliferation of white pulp of spleen into hemosiderin bodies, particularly where no antibodies were produced (complete immunosuppression).[245] Panigrahi and Misra[246] determined the extent of physiological damage caused by $Hg(NO_3)_2$ on *Tilapia mossambica* exposed for 35 d. A concentration of 0.5 mg/l caused blindness, swelling of the eyes, and decreased O_2 consumption. Oxygen uptake rate increased to 74% of the initial value when fish were transferred to clean water. Hematological effects included decreased protein content, hemoglobin percentage, and RBC count and microhematocrit percentage. Mercury was accumulated more in the liver than in the brain and muscle, and was depleted more rapidly in liver when fish were transferred to clean water.

Becker and Wolford[247] exposed rainbow trout to Ni at various temperatures. It was found that Ni significantly decreased the thermal resistance to increasing temperatures at 1.5 mg Ni^{++}/l but not at 0.9 mg Ni^{++}/l after 28 of Ni exposure. Temperature resistance of coho salmon juveniles was not significantly altered at a lethal level of 11.2 mg Ni^{++}/l. Tomasso *et al.*[248] compared the inhibitory effects of $CaCl_2$ and NaCl on nitrite-induced mortality and methemoglobinemia in channel catfish. Methemoglobin levels in fish exposed to 2.5 mg nitrite/l and up to 30 mg chloride/l as either $CaCl_2$ or NaCl were similar but significantly lower than controls. However, higher chloride levels (60 mg/l) with up to 10 mg nitrite/l did not result in significant decreases from the control. When lower levels of chloride were used (15 mg/l), fish were more protected by NaCl than in exposures with $CaCl_2$. Chitra and Rao[249] determined the effect of NaF on mortality, physiological variations, and hematological parameters of *Channa punctatus*. A concentration of 10 mg NaF/l decreased O_2 consumption, hematocrit values, total leucocyte count, percentage of monocytes and lymphocytes, and liver somatic index but increased erythrocyte count, RBC size, MCHC, MCV, and mean corpuscular hemoglobin. Variation in response of the sexes of these fish to treatment was observed. The response of the tench (*Tinca tinca*) to KNO_3-enriched water was assessed by Demaël *et al.*[250] A concentration of 8.5 mg KNO_3/l increased cortisol and glucose levels after 35 d. Free fatty acids and glycerol also increased in the plasma but became normal by Day 20. The ion distribution in erythrocytes and liver increased relatively early, there was no observable change in water content and glycogen levels varied in tissues studied. Acute and chronic exposures of rainbow trout to Se(IV) as sodium selenite were conducted by Hodson *et al.*[251] Exposure to sublethal Se concentrations from the egg stage through 44-wk post-hatch resulted in increased mortality of eyed eggs at concentrations $\geq$ 28 μg Se/l, decreased red blood cell volumes at $\geq$53 μg Se/l, and decreased cellular blood iron at $\geq$16 μg Se/l. Tissue analyses showed the highest Se residue accumulations in the liver, digestive tract, and kidney. Kelly *et al.*[252] perfused the gills of eel from fresh and salt water with orthovanadate in Ringers solution. Microscopic examination showed vasoconstriction in the secondary gill filaments that may have had an effect on osmoregulation. Sargent *et al.*[253] reported that intravenous injection of 23 μg V as $Na_3VO_4 \cdot 14H_2O$ into rainbow trout significantly slowed fish heart rate. Exposure of trout to orthovanadate in water did not alter heart rate nor gill ventilation rate. The effect of concentrations of 5, 10, 15, and 20 mg Zn/l (as $ZnSO_4$) on levels of serum glucose and lactate were studied in nine nutritionally important tropical fish species: *Cirrhima mrigal*, *Labeo rohita*, *Catla catla*, *Ophicephalus punctatus*, *Ophicephalus striatus*, *Ophicephalus marulius*, *Clarius batrachus*, *Heteropneustes fossilis*, and *Mystus seenghala*.[254] In all nine species an inverse relationship was observed between the concentration of Zn and the accumulation of serum glucose and lactate. Mishra and Srivastava[255] investigated the hematological changes that occurred in freshwater teleost (*Colisa fasciatus*) after 90 h of exposure to 100 mg Zn/l. Total red cell counts, hematocrits, total white cell counts, and the number of small lymphocyte cells decreased significantly, whereas erythrocyte sedimentation rate increased markedly from the controls. A significantly higher hepatosomatic index was recorded for Zn-treated fish than for unexposed animals. The effects of Zn on LDH, MDH, and alkaline phosphatase from cultures of fathead minnow cells were examined by Adragna and Privitera.[256] Both LDH and MDH (on a per cell basis) activity increased with increased Zn concentrations in the medium whereas alkaline phosphatase activity decreased. Watson and Beamish[257] examined effects of Zn on branchial ATPase activity *in vivo* in rainbow trout. ATPase activity was generally stimulated by Zn after 30 d but levels were not appreciably altered at higher Zn levels of 0.99 and 1.98 mg/l. Enzyme activity appeared to increase with increased temperature. Katz[258] conducted a series of acute exposures of the mosquitofish to various toxicants, such as metals, Cl_2, and an osmotic sugar solution to measure changes in Na flux from the fish. It was concluded

that much of the difference in toxic effects levels was caused by differential gill permeability. Hughes *et al.*[259] exposed rainbow trout to Ni and Cr at concentrations of 0.05 and 0.2, respectively, of the 48-h LC_{50} for 3.5 days and to Cd at a concentration of 0.001 of the 48-h LC_{50} for 234 d. The authors made morphometric measurements of structural components of the gill secondary lamellae and calculated diffusing capacities. Measurements of diffusing capacities indicated changes in the ability of the fish to transfer O_2 from water to blood. Nickel appeared to have a harmful effect, whereas only trends were apparent for Cr and Cd.

Behavioral changes in bluegills that were often related to the social rank of fish were observed[260] upon exposure to a mixture of $CdCl_2$ and $ZnCl_2$. Studies on the effects of Li on social aggregation in the goldfish showed that aggregation was reduced between two fish that had received Li treatments of 10 to 30 mmole/l.[261] When Li-treated fish were investigated in groups of three, maximal disruption occurred but only partial social disruption occurred when two out of three fish were treated. Fisher *et al.*[262] assessed the effects of hydrazine (N_2H_4) on bluegill in terms of acute toxicity and behavioral dorsal light responses and aggressiveness. Dorsal light response in the presence of artificial prey was significantly decreased within 15 min of exposure of hydrazine concentrations well below the 96-h LC_{50}. Aggressiveness as measured by the number of attacks on an artificial prey increased in a dose-related manner. Avoidance response bioassays with juveniles of largemouth bass, bluegill, and rainbow trout using eight chemicals were conducted by Black and Birge.[263] Avoidance was significant with Cd, phenol, and Zn; attraction was significant with chloroform, dioctyl phthalate, and Hg; and NTA and Cu produced variable responses.

Organics. Olson and Christensen[264] determined the effects of 74 chemicals of various classes on the activity of acetylcholinesterase preparations (*in vitro*) from the muscle of the fathead minnow. A highly inhibitory effect was found with several cabamates, one organo-oxy-phosphate, the arsenite ion, and certain heavy metal cations. Several organochloride pesticides, alkaline and alkaline-earth cations, anoins, drugs, and other chemiçals had little inhibitory effect at the highest concentration, 0.01 M. In screening tests conducted on thousands of organic and inorganic compounds, Kenaga and Moolenaar[265] compared the relative toxicities of the chemicals to daphnia, fish, aquatic plants, and algae. Emerald shiner (*Notropis atherinoides*), fathead minnow, goldfish, and carp were tested in about equal frequency. It was concluded that the animals were generally more sensitive indicators of acute toxicity than plants and, therefore, water quality limitations based on toxicity data for fish and daphnids should also protect algae and aquatic vascular plants. Reiff *et al.*[266] compared the ability of various toxicity test methods used in the United Kingdom to measure the toxicity of 11 detergents. Neither the method nor the species affected the results more than an order of magnitude (defined as the range of values between the average multiplied or divided by the square root of 10). These tests compared favorably to those performed by several German laboratories using a single method. Paasivirta *et al.*[267] measured the enrichment of several chlorinated phenolic compounds (2,4,6-trichlorophenol; 2,3,4,6-tetrachlorophenol; 4,-5,6-trichloroguaiacol; pentachlorophenol; tetrachloroguaiacol; and tetrachlorocatechol) in food chain organisms in three lakes in middle Finland. Concentrations in pike and roach (*Rutilus*) ranged between 1 and 100 ng/g. Some of these compounds, most clearly tetrachloroguaiacol, were enriched in the food chain. In order to evaluate a continuous-flow procedure for evaluating effects of insoluble and volatile organic compounds on embryo-larval stage of fish, Birge *et al.*[268] selected test compounds covering a range of solubility and volatility. Chlorinated aromatic hydrocarbons were found to be the most toxic compounds, followed closely by chloroform and phenol. It was concluded that when exposure was maintained from fertilization through 4 days' post-hatch and responses for lethality and teratogenesis were combined, LC_1 values from a log probit analysis provided a close approximation to MATCs determined in partial and complete life cycle tests. A hyperbolic model of time-dose interations for mortality of fish exposed to naphthalene was developed by Taylor[269] and tested on the mummichog, *Fundulus heteroclitus*. Mortality data were used to calculate the dose for which appearance of the response would require an infinite exposure time. Of toxicity indicators examined, only sensory cell necrosis was a more sensitive indicator of toxicity than mortality. A mathematical model of PCB accumulation in Lake Ontario was developed[270] in which organisms in the food chain were analyzed along a continuum according to size. The model was applied to PCB data from Lake Ontario and indicated that introduction of organism size as an independent variable in the mass balance of a toxicant provided a generalized analysis framework. The model permitted the integrated use of diverse laboratory experiments on uptake and excretion as well as an interpretive framework for field data of toxicant concentrations. Hunder *et al.*[271] surveyed the accumulation of PCBs in sediments and fish of an Oklahoma stream contaminated from an industrial source. Predators were represented by white bass (*Morone chrysops*) and white crappie, omnivores by channel catfish, and detritivores by the river carpsucker (*Carpiodes carpio*) and carp. The concentration of PCBs in fish fillets from the detritivores were significantly greater than in the omnivores and predators, and there was no significant difference between the latter two. The accumulation of PCBs in the American shad (*Alosa sapidissima*) was measured at the time of their migration in the Hudson River during the spring of 1977.[272] Because these fish do not eat in freshwater before spawning, it was assumed that any contamination would have entered their bodies directly from the water. PCB levels in the fish ranged from 2.0 μg/g at a site 27 miles from the river mouth to 6.1 μg/g at a site 75 miles upstream from the river mouth. Merna[273] assessed the extent of contamination of stream fish and streams from the death and decay of coho and chinook salmon containing residues of PCB and DDT. Brown trout, and to a lesser extent mottled sculpins (*Cottus bairdi*) and slimy sculpins (*Cottus cognatus*), accumulated PCB and DDT as a result of eating salmon eggs that ranged from 0.46 to 9.50 μg PCB/g. Concentrations of chlorinated hydrocarbons in trout fillets ranged from 0.20 to 4.63 μg PCB/g and 0.11 to 2.52 μg DDT/g. In a survey of hepatomas in Atlantic tomcod (*Microgadus tomcod*) collected from the Hudson River estuary in New York, Smith *et al.*[274] found that 20% of the fish had livers with neoplastic nodules and hepatocellular carcinoma. The exact cause of the high prevalence of hepatocellular carcinoma was unknown but PCBs were suspected of having a role. Cairns and Parfitt[275] presented data on the presence of TDE and its

metabolites, DDMS, DDNU, and DDNS, in fish from Clear Lake (Calif.). The persistence of parent TDE after 19 y in the lake was clearly demonstrated. The highest levels of TDE were observed in the carp.

A number of pathological conditions (that is, disease, deformity) were found to correlate directly with pollution load in three diverse watersheds.[276] It was found as the pollution load resulting from the presence of organochlorine and organophosphate chemicals increased, more fish were observed with disease problems. A muddy flavor and odor in commercial walleye, lake herring (*Coregonus artedii*), northern pike, and lake whitefish from Cedar Lake (Manitoba), Canada were found by Yurkowski and Tabachek[277] to result from the presence of geosmin and 2-methylisoborneol, and these are known to cause a muddy odor in fish. The source of these compounds was not established, but they are known to be produced by blue-green algae and actimomycetes. In a study of the occurrence of organohalogens in pristine European and Antarctic aquatic environments, Ballschmiter and Zell[278] examined organochlorines in Arctic char (*Salvelinus alpinus*) from the Tyrolean Alps, Antarctic cod, anchovy, and sperm whale oil. The Arctic char sample, where only an input by air should be possible, showed consistently the highest value.

Stay *et al.*[279] studied the effects of forest fertilization with urea on the biota of cascade streams in Oregon. No mortalities in rainbow trout were found that could be attributed to byproducts or contaminants of urea during a 2-mo exposure. A study of acute toxicity of N-nitrosodimethylamine and N-nitrosodiethylamine to aquatic life[280] showed that invertebrates and fathead minnows were relatively insensitive compared to algae. The 96-h LC_{50} values for fathead minnows were near 1 000 mg/l for both compounds, about 1 000 times higher than for algae. In an evaluation of the ecological effects of water-soluble chlorinated organic compounds of low volatility, Trabalka and Burch[281] measured the toxicity of 5-chlorouracil and 4-chloroesorcinol to carp embryos. Earlier results that indicated that these compounds were toxic to carp embryos at concentrations much less than 1 mg/l were refuted. Rather, a low to moderate toxicity (LC_{50} greater than 10 to 100 mg/l) was indicated for these compounds. Madeley and Birtley[282] investigated acute toxicity and bioaccumulation of chlorinated paraffins (CPs), represented by Cereclor 42, to rainbow trout. Up to 96 hours, CP emulsions were nontoxic to rainbow trout at concentrations ranging from 520 to 1 630 mg/l. No sublethal effects because of the opacity of the emulsions were observed. The concentration of CP in both liver and gut approached that administered in the food, whereas other tissues showed less uptake. Stalling *et al.*[283] measured the toxicity of PCB components to rainbow trout in order to determine if differences in PCB toxicity were related to o,o'-Cl substitution. No major difference in toxicity below 8 μg/l was observed in fish exposed to similar concentrations of 1–4 o,o'-Cl fractions of Aroclor 1248. However, exposure to all concentrations of the non-o,o'-Cl substituted 3,4,3'4'-tetrachlorobiphenyl resulted in poor growth, reduced food consumption, and disorientation in test fish. In a study of the effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on the early life stages of the pike, Helder[284] exposed fertilized pike eggs to TCDD concentrations between 0.1 and 10 ng/l for 96 h. At all concentrations, egg development was retarded and fry growth was significantly retarded for a long period after exposure. A dose-related mortality was observed, as well as various histopathological effects. Stoss and Haines[285] examined the effects of toluene on embryos and fry of Japanese medaka (*Oryzias latipes*). Acute toxicity was determined and concentrations as low as 41 mg/l produced teratogenic effects in susceptible embryos. A method for rapid estimation of the MATC based on observable effects on medaka eggs was proposed. Moles[286] examined the sensitivity of coho salmon fry, parasitized by glochidia of the freshwater mussel *Anodonta oregonensis*, to toluene, naphthalene, and crude oil. Fry infested with 20 to 35 glochidia and above were significantly more sensitive to each of the toxicants than uninfested fish. Sensitivity increased linearly with increased parasite numbers. The short-term lethal concentration and the long-term effect of waste oil on the survival, growth, and reproduction of the American flagfish (*Jordanella floridae*) was determined by Hedtke and Puglisi.[287] Tissue residue analyses indicated uptake of Pb, Cd, and Zn at higher exposure levels. Based on observed uptake, compounds observed in the water-soluble fraction, and previous toxicity data, it was speculated that these metals probably contributed significantly to the toxicity of the waste oil tested. Doe and Wells[288] measured the acute toxicity and dispersing effectiveness of 18 oil spill dispersants. Rainbow trout in fresh water were more sensitive than the two marine fish tested, *Fundulus heteroclitus* and *Menidia menidia*, whereas fourth-stage larval lobsters (*Homarus americanus*) were least sensitive.

Holcombe *et al.*[289] observed that toxic effects produced by short-term exposure of fathead minnows to 2,4,-dichlorophenol were reduced when the pH of the test water was increased from approximately 7.5 to 9.0 by the addition of NaOH. Sodium chloride at 1 to 13.9 mg/l had no observable effects on acute toxicity of 2,4-dichlorophenol.

Kobayashi and Kishino[290] determined the effect of pH on the toxicity and accumulation of pentachlorophenol in goldfish. Both toxicity and accumulation were reduced at higher pH (6 to 10) with respect to total PCP. It was assumed that the reduced activity was because of a shift in chemical equilibrium between undissociated and dissociated forms with increasing pH. The effect of a bacterial infection on the toxicity of sodium pentachlorophenate (NaPCP) to juvenile coho salmon was investigated by Iwama and Greer.[291] As determined by toxicity tests, the susceptibility of the infected coho salmon to NaPCP was significantly greater than for uninfected coho salmon. DeGraeve *et al.*[292] measured the acute and embryo-larval toxicity of several phenolic compounds to rainbow trout, fathead minnow, and *Daphnia*. In flow-through bioassays, 96-h LC_{50} values for rainbow trout and fathead minnows ranged from less than 0.1 mg/l for hydroquinone to greater than 100 mg/l for resorcinol. For both species, the embryo-larval no-effect concentration for phenol was approximately 1% of the 96-h LC_{50}. Hirose and Yasuno[293] determined acute toxicity of three molluscicides to juvenile guppy. Juveniles were separated into two groups soon after parturition; one group was fed and the other group was not fed. The 24-h LC_{50} value of B-2 (sodium 2,5-dichloro-4-bromophenol), the least toxic of the molluscicides, did not differ until 2 days after parturition. The 24-h LC_{50} value of the starved group did not change up to 10 days, whereas the fed group became more resistant with time (up to 18 days). Yurimin (3,5-dibromo-4-hydroxy-4'-nitroazobenzene) was the least toxic of the com-

pounds. The 24-h LC_{50} values of niclosamide (5,2'-dichloro-4-nitrosalicylicamide) to fed juveniles decreased linearly with the age of the fish. As part of a study of the toxicity of metabolites of MCPA (4-chloro-2-phenoxyacetic acid), Hattula *et al.*[294] measured the acute toxicity of 4-chloro-o-cresol to brown trout. Liver, kidney, and gills were also examined by light microscopy for histopathological changes, and tissue concentrations of cresol were determined. The results indicated that cresol had a greater bioconcentration potential and caused more extensive histological changes than the parent compound. Batcheld *et al.*[295] determined the toxicity of 14 members of the versene family of chelating agents to bluegill sunfish. Acute toxicity (96-h LC_{50}) ranged from 159 to 14 100 mg/l. The effect of water hardness on toxicity was also investigated for three versenes. The acute toxicity of an irritant compound known as "CS gas" (o-chlorobenzyli-denemalonotrile) and of its two principal hydrolysis products to rainbow trout was determined by Abram and Wildon.[296] The by-products, o-chlorobenzal-dehyde and malononitrile, appeared to behave synergistically. The toxicity of commercial non-ionic surfactant Imbentin-N/52 (etho-nonylphenol) and two non-ionic antifoam agents Paracum-21/SM (ethoxylated carbonic acids) and Paracum-ASD (mixture) to rainbow trout was determined by Müller.[297] The addition of antifoam agents to surfactant solutions further lowered surface tension but did not substantially alter the fish toxicity of the surfactant. No fish died or manifested a response in the antifoam agents alone between 5 and 500 mg/l. Maki[298] examined the use of respiratory activity in bluegill sunfish as a predictor of chronic fish toxicity values for the surfactants. Good correlations were observed between reported MATCs for fathead minnow and the concentrations of surfactants that elicited statistically significant changes in the diurnal ventilation frequencies of exposed bluegills. An apparent acclimation of test fish between 47 and 72 h of exposure was noted. Holman and Macek[299] conducted acute and life cycle chronic toxicity tests on fathead minnow with three commercial linear alkyl-benzenesulfonates (LAS). The no-observed-effect concentration depended on the mean alkyl chain length of the LAS. Embryo and larval stages of development were consistently the most susceptible.

Southworth *et al.*[300] investigated the potential for a mutagenic azaarene, acridine, found in synthetic fuel effluents to accumulate in the fathead minnow by four possible pathways: direct uptake from water, uptake via contaminated sediments, uptake via ingestion of contaminated zooplankton, and uptake via ingestion of benthic invertebrates from contaminated sediments. The calculated rates of uptake of acridine via ingestion of contaminated invertebrates and ingestion of sediment were negligible compared with direct uptake from water. Uptake, metabolism, and elimination of radiolabeled 1,2,4-trichlorobenzene in fingerling rainbow trout and carp were measured by Melancon and Lech.[301] For 2 d following an 8-h exposure, the elimination half-time of ^{14}C from muscle and liver was 0.4 d, but following a 35-d exposure, an early elimination half-time of 0.4 d was followed after 4 d by a slower elimination half-time of 50 d. Larger trout and carp were exposed to obtain enough material for biotransformation studies. Residues of chlorinated benzene in trout (*Salmo trutta*), nase (*Chondrostoma nasus*), and whiting (*Leuciscus cephalus*) from Slovenia were generally well below 0.1 μg/g. The exception was 1,4- and 1,3-dichlorobenzene with maximum detected levels of up to 0.45 and 1.20 μg/g, respectively.[302] Macek *et al.*[303] evaluated the relative importance of dietary and aqueous exposure of bluegill sunfish to di(2-ethylhexyl)phthalate, 1,2,4-trichlorobenzene, and leptophos. Results indicated that the incremental steady-state body burden resulting from dietary exposure was statistically indistinguishable from that following aqueous exposure. Critical analyses of similar studies with Cd, endrin, and Kepone supported the hypothesis. DDT appeared to be the only chemical of those reviewed with a potential for significant accumulation of residues through an aquatic food chain. Kinetics of accumulation and elimination in female guppies of six chlorobenzenes ranging from dichlorobenzene to hexachlorobenzene (HCB) were studied by Könemann and Van Leeuwen.[304] BCFs were calculated and ranged from 10 to 10^3 for dichlorobenzene and to 10^5 for hexachlorobenzene. Niimi and Cho[305] monitored HCB residues in rainbow trout fed prepared diets that contained HCB at concentrations in the low μg/kg range, approximating levels of lower trophic levels in Lake Ontario. Over a range of HCB in the diet of 4 to 780 μg/kg, HCB residues in the fish leveled off after 57 d to approximately that in the food. Studies on the accumulation, elimination, and metabolism of 3,3'-dichlorobenzidine (DCB) in bluegill sunfish[306] showed that DCB residues were distributed in both edible and non-edible portions and that the parent compound and its metabolites were not completely eliminated upon transfer of the fish to water free of DCB. The only metabolite detected appeared to be an N-glucuronide.

Branson *et al.*[307] determined the taint threshold of diphenyl oxide (DPO) in rainbow trout. A statistically significant correlation was obtained between fish-taint scores and the DPO concentration in flesh. Residues of hexazinone (active ingredient of Velpar weed killer) in bluegill sunfish over a 4-wk exposure period peaked at 2 weeks and then declined through the remainder of the exposure.[308] After 1 week in non-contaminated water, residues declined by greater than 90% and no detectable residue remained after 2 weeks following exposure. No effects on the fish were observed throughout the test. The relative disposition between brain, liver, and blood of orally administered naphthalene and its metabolites in rainbow trout was investigated by Collier *et al.*[309] Naphthalene concentrations in brain approximated those found at the same time in liver. A non-conjugated naphthalene derivative also accumulated in the brain, to a lesser degree than in liver, but conjugated naphthalene derivatives were largely excluded from the brain. These findings suggested that the brain of adult trout was substantially different from other tissues (liver and blood) with respect to disposition of naphthalene and its metabolites. Studies of uptake, disposition, and elimination of 2-methylnaphthalene and naphthalene in rainbow trout, bluegill sunfish, and carp indicated that elimination rates of ^{14}C were a composite of the rates of the parent compound and its metabolites.[310] Conjugates of these metabolites in the bile were present at levels of more than 100 000 times the water level of the parent chemical, supporting the use of bile sampling for monitoring environmental pollutants. Woidich *et al.*[311] examined PCBs and other chlorinated hydrocarbon content in trout (species undefined). PCBs ranging from trichlorobiphenyls to hexachlorobiphenyls, DDE, DDD, DDT, and chlordane were found and identified by mass spectrometry. It was concluded that fewer chlorinated PCBs were not deposited or rapidly excreted. Distribution and elimination of PCBs during

the egg, sac-fry, and fry stages of rainbow trout were studied by Guiney *et al.*[312] The majority of PCB in eggs were associated with yolk fluid. During sac-fry stage, PCB was primarily in yolk sac but was gradually transferred to larva as the yolk sac was absorbed. During the egg and first two-thirds of the sac-fry stage, PCB elimination was low, but the half-life decreased from 231 d to 15 d during the last third of the sac-fry stage and into the fry stage. Shea *et al.*[313] measured the biological availability of PCB in soil sediment to goldfish and determined whether activated carbon would inactivate PCB in soil and prevent its bioaccumulation. It was found that active carbon was 90 to 100% effective in reducing the bioavailability of PCB, as measured by accumulation in muscle tissue, and that accumulation during 5-d exposure periods decreased with time after preparation of the system, possibly because of settling of contaminated sediment.

The relation between chemical structure of phenols and their elimination by goldfish, following intraperitoneal administration, was investigated by Nagel and Urich.[314] Based on four compounds (phenol, 3-nitrophenol, 3,5-diethylphenol, and 4-aminophenol) it was concluded that phenols with increased polarity and decreased molecular weight could be more effectively eliminated across the gills. The relative importance of brachial, renal, and biliary excretion routes of PCP in goldfish was determined by Kobayashi and Nakamura.[315] The amounts of PCP excreted by way of these three routes corresponded to 52, 24, and 22% of the total for 24 h after a 24-h exposure to 0.1 mg PCP/l. Excretion from the body surface was negligible in comparison. Approximately 30% of the PCP excreted from the gills was in a free form, whereas almost all in the bile and urine was in a conjugated form. Experiments on uptake, distribution, and elimination of two sodium alkylpoly (oxyethylene) sulfate surfactants in carp showed that these compounds were first rapidly absorbed in gills and skin and then transferred in other tissues and organs.[316] Along with their metabolites, they were eliminated rapidly from gills, blood, skin, hepatopancreas, and kidney; moderately rapidly from spleen, brain, gonad, and muscle; and slowly from gall bladder and intestinal content.

Bioconcentration and metabolism of LAS by fathead minnows was measured by Comotto *et al.*[317] Uptake was a function of exposure concentration and chain length. In depuration studies, greater than 85% of the radio-labeled compound was cleared from all tissue within 3 to 4 d with nearly 100% clearance within 10 d. Sako *et al.*[318] investigated the pharmacodynamics of trimethoprim (TMP) in the ayu (*Plecoglossus altivelis*) after oral administration of TMP-sulfadoxine mixture for control of vibriosis. It was concluded that TMP was retained in tissues for a matter of hours and was completely excreted within 1 d. A study of the uptake of toluene in bluegill sunfish and crayfish[319] showed that toluene concentration in gill, ovary, heart, liver, kidney, and brain remained essentially constant, or increased slowly, over a 48-h test period. The concentration of toluene in the spleen, gall bladder, gut, and testes increased by a factor of 2 or greater during the experiments. A steady increase in toluene concentration in liver concomitant with a large accumulation of radiolabeled material in the gall bladder suggested that bluegills may metabolize toluene in the liver and store the metabolites in the gall bladder. In a related study,[320] the accumulation of toluene in bluegill sunfish that fed on toluene-contaminated mosquito larvae indicated that with the exception of the stomach and intestine there was no difference in the concentration of toluene in the organs of experimental and control fish. Neely[321] proposed a method for estimating, in the absence of experimental data, the rate constants for the uptake and clearance of chemicals by fish. Rate constants were based on physiology of fish and properties of the chemical. Model credibility was demonstrated by comparing predicted rate constants with values for DEHP, chlorpyrifos, and TCDD measured in the laboratory.

Zaleski *et al.*[322] determined the influence of aflatoxin B_1 (AFB_1) on the survival time of rainbow trout (*Salmo gairdneri*) brood. Complete mortality was observed in 84 d with fish fed food containing 0.060 μg AFB_1/g and in 90 d with 0.045 μg AFB_1/g. Fish fed 0.030 μg/g and the controls experienced 28% and 60% survival, respectively, after 90 d. An investigation of the carcinogenic response of rainbow trout to aflatoxin Q_1 (AFQ_1)[323] showed that AFQ_1 fed at 100 ng/g in the diet for 10 months was a potent liver carcinogen but was approximately 100 times less active than AFB_1. Administration of 50 μg/g of methyl sterculate, a cyclopropenoid fatty acid, in combination with 100 ng AFQ_1/g resulted in a synergistic tumor response. Goel and Garg[324] found that following the exposure of *Channa punctatus* to the azo dye 2,4-diamino-3-aminoazobenzene there was a marked fall in the red blood cell count, hematocrit value, and hemoglobin content. Biochemical analysis of plasma revealed a significant decrease in total proteins and sugar associated with an increase in cholesterol, urea, nitrogen, creatinine, Ca, and Mg in treated fishes. Investigations of the action of PCBs on the hepatopancreas lysosomal membranes of carp were carried out by Ito *et al.*[325] Results suggested that PCBs may induce instability of the lysosomal membrane, depending on the lipophilic nature of PCB. In other studies by Ito and Murata,[326] carp were fed a diet containing 250 μg/g PCB (Aroclor 1248). Following the exposure, serum glucose content increased significantly and there were also significant increases in the activities of several serum enzymes. Changes in other enzymes were either not significant or negative. The effects of several organochlorine compounds on interrenal activity and cortisol metabolism in *Tilapia aurea* were measured by Ilan and Yaron.[327] Cortisol output from superfused interrenal tissue, in response to a pulse of procine ACTH, was measured by radioimmunoassay. Addition of PCB or p,p-DDT to superfused medium (at 50 mg/l) did not alter cortisol output from the tissue in response to ACTH. At a concentration of 100 mg/l p,p,'-DDE inhibited this response, whereas, o,p'-DDD proved effective even at a concentration of 1 mg/l. Structural changes of intestinal mucosal cells of juvenile chinook salmon exposed in parallel experiments to petroleum hydrocarbons and chlorinated biphenyls were studied by Hawkes *et al.*[328] using light and electron microscopy. The mucosa was intact in control and petroleum hydrocarbon-exposed fish, but some exfoliation was observed in the fish exposed to chlorinated biphenyls. A group fed the combined contaminants had considerably increased sloughing, indicating an interactive effect. The effect of an i.p. treatment of CCl_4 on the urine flow rate of rainbow trout[329] showed that an oliguria or anuria was observed as early as 1-h post-treatment, as well as a significant reduction in the 24-h urine output. Early oliguria and lack of demonstrable and histopathological changes suggested that reduction in urine flow rate was not

the result of a direct toxic effect on the kidney. Histological studies on toxicity of phenol and CCl_4 to liver and kidney of common carp and Crucian carp (*Cyprinus auratus*) were made by Lifan and Ruitao.[330] Fish were exposed to phenol in water, whereas CCl_4 was injected into the coelomic cavity. Both compounds resulted in the formation of a number of vacuoles, enlargement of nuclei, and deformation of the nuclear membrane in some liver cells. No histological changes were observed in liver tissue. Effects of phenol and dinitrophenol on acid and alkaline phosphatases in tissues of the Indian freshwater fish, *Notopterus notopterus*, were investigated by Verma *et al.*[331] Individually and in combination, the two compounds inhibited the activity of both enzymes in the kidney, heart, brain, gills, muscles, stomach, intestine, and pyloric caeca. Dinitrophenol was more inhibitive than phenol, and the two together acted synergistically. Schnell *et al.*[332] investigated the mono-oxygenase activities of coho salmon liver microsomes toward three polycyclic aromatic hydrocarbon substrates, benzo[a]pyrene, 2,6-dimethylnaphthalene, and naphthalene. The results indicated that the microsomal preparation had a high affinity for benzo[a]pyrene, intermediate affinity for 2,6-dimethylnaphthalene, and a low affinity for naphthalene.

The Indian freshwater Sauli (*Channa punctatus*) was exposed by Verma *et al.*[333] to two synthetic detergents, Idet 5L and Swanic 6L, and the effects of those compounds on the ATPase system in the brain and gill were determined. Enzyme inhibitions were highest in oligomycin-insensitive Mg^{++} ATPase. The mechanism of action was postulated to be related to ability of these compounds to alter the cellular membrane configuration by binding with lipid portion of the membrane. Bolis and Rankin[334] studied interactions between the vascular actions of an LAS detergent and catecholamines in perfused gills of European eel and brown trout. Gills in fish kept for 24 or 48 h in water containing 1 mg/l of LAS showed greatly reduced responses to noradrenaline. Inhibition tended to be greater in trout than in eel, and noradrenaline action was almost completely abolished in trout exposed for 48 h. It was concluded that chronic exposure to detergents may restrict the ability of fish to dilate their gills. Teller *et al.*[335] determined the effect of non-ionic surfactant Triton X-100 on cytochrome oxidase activity in carp, frog, and rat liver mitochondria. It was observed that carp mitochondria were less susceptible to Triton X-100 than were frog or rat mitochondria. An investigation of the carcinogenicity of N-methyl-N′-nitro-N-nitrosoquanidine (MNNG) to rainbow trout[336] indicated that a single short exposure of 21-day-old embryos in the ambient water was sufficient to induce, in a dose-dependent fashion, a high incidence of hepatocellular carcinomas and lower incidence of nephroblastomas within 9 to 12 months. A marked sex reversal (female to male) trend was also noted in the group exposed to 100 gm MNNG/l.

The effects of pentobarbital sodium on intraspecific attack behavior in male Siamese fighting fish (*Betta spendens*) were investigated by Figler and Klauenberg[337] in an attempt to extend earlier findings with chlordiazepoxide and secobarbital sodium. Fish exposed to 40 μg/ml pentobarbital sodium showed significantly less attack behavior than control fish and fish exposed to 20 μg/ml. Studies on effects of antitranspirant Folicote on aquatic and terrestrial life by McMahon and Kynard[338] investigated the ability of western mosquitofish to detect and avoid the chemical in a Y-maze. Test fish avoided Folicote concentrations greater than 0.75%, indicating mosquitofish could avoid potentially lethal conditions resulting from Folicote use.

INDUSTRIAL AND MUNICIPAL EFFLUENTS

Chlorine and Wastewater. Avoidance tests with chlorine and six species: carp (*Cyprinus carpio*), common shiner (*Notropus cornutus*), spotted bass (*Micropterus punctatus*), white sucker (*Catostomus commersoni*), channel catfish (*Ictalurus punctatus*), and coho salmon (*Oncorhynchus kisutch*), were conducted by Cherry *et al.*[339] All fish species demonstrated an active avoidance at levels ≤ 0.41 and 0.27 mg/l total residual chlorine (TRC) and free residual chlorine (FRC), respectively. In most instances, avoidance occurred at 0.20 mg/l TRC (0.15 mg/l FRC) or lower. Ward and DeGraeve[340] reported that the acute 96-h toxicity values for nine tested fish species ranged from 0.029 mg Cl_2/l for the pugnose minnow (*Notropis anogenus*) to 0.519 mg Cl_2/l for the goldfish (*Carassius auratus*) Ozone, SO_2, and chlorobromine were less toxic. Elmore *et al.*[341] found that pulsed additions of monochloramines had a 96-h lethal threshold of 1.25 mg/l on young goldfish and a 96-h LC_{50} value of 0.80 mg/l was determined mathematically. Temperature increases resulted in increased sensitivity to chloramine. Striped bass (*Morone saxatilis*) were tested for chlorine sensitivity with regard to the effect of test temperature, exposure concentration, and exposure length.[342] Eggs were only affected by chlorine concentration and exposure time, whereas newly hatched fry were affected by all three test conditions. Striped bass were found to be affected most by chlorine concentration, exposure duration, and temperature change in descending order.[343] A predictive scheme of all factors for use by power plant operators was presented. Yosha and Cohen[344] studied the effects of intermittent chlorination on the zebrafish (*Brachydanio rerio*). It was found that unhatched embryos could resist chlorine levels of 1 mg Cl_2/l and newly hatched larvae had an LC_{50} of approximately 0.75 mg Cl_2/l. The most toxic situation was at low ionic strength in test water. Schumacher and Ney[345] indicated the avoidance reaction of rainbow trout (*Salmo gairdneri*) to TRC was apparent at a concentration as low as 0.05 mg TRC/l. Larvae of white perch (*Morone americana*) were exposed to total chlorine to determine its effects in combination with various temperatures and exposure times.[346] Only chlorine concentration and exposure time had an effect on survival of white perch. Hall *et al.*[347] performed temperature and chlorine multiple stress studies with a white perch to simulate exposure to power plant effluents. The fish were found to have delayed reaction to such exposures for up to 36 h after the initial exposures. A predictive model was developed and showed that short intermittent high chlorine exposures or rapid mixing into the receiving water for low continuous exposures were safest. Cairns and Conn[348] studied the effect of chlorine, ozone, chlorine dioxide, and dechlorinated effluents on rainbow trout. Chlorine and chlorine dioxide were found to be toxic between 0.03 to 0.09 mg/l and 35 to 45 mg/l, respectively, whereas other materials exhibited no toxicity under test conditions. Tsai and McKee[349] measured acute toxicity of mixtures of chloramines, Cu, and LAS de-

tergent to goldfish. It was found that the individual toxicity of these chemicals was either additive or synergistic in mixtures, depending on rate of toxic action of the individual chemical, the toxicity ratio of chemicals in the mixtures, and the concentration of mixtures. Hoke and Prater[350] tested elutriates from five lake harbor sediments on *Pimephales promelas* and several invertebrate species. They reported that eluted Cl^- ion correlated best with toxicity to the fish with a release level of 41 mg Cl^-/l being the apparent threshold.

Domestic and industrial wastewaters in Germany were tested by Becker-Birck and Havemeister[351] to determine toxicity to orfe (*Leuciscus idus melanotus*). Domestic wastewater from secondary treatment proved to be nontoxic in 50% of those tested. Industrial effluents resulted in maximal LD_{50} concentrations of 62.5 g/l. Observations and experiences with the methods of the orfe test were discussed. Cairns *et al.*[352] used fish toxicity tests with various European species to evaluate the toxicity of various fractions of effluent from a German chemical complex. Data revealed that even after treatment by activated sludge and other conventional processes, effluent was lethally toxic to rainbow trout. Fathead minnows (*Pimephalas promelas*) and eggs and fingerlings of rainbow trout were exposed to an oil-shale process water.[353] Fish were exposed to a simulated wastewater containing 13 of the major constituents of the waste. Results of 96-h bioassays were very similar: 0.56 to 1.12% of the waste and simulated waste, respectively, for fathead minnows, and 0.41 and 0.56% for rainbow trout. Sublethal effects of Omega-9 water and an *in situ* oil shale retort water on rainbow trout were determined by Lebsack *et al.*[354] Exposure for 96 h to 0.3% Omega-9 water (70% of the previously reported LC_{50}) decreased blood packed cell volume, hemoglobin concentration, and plasma alkaline phosphatase activity. No sublethal effects on these parameters occurred at levels less than 20% of the LC_{50}. Walsh *et al.*[355] exposed several freshwater and marine organisms to textile mill effluents. Generally the fishes were less sensitive to wastes than crustaceans based on LC_{50} and EC_{50} data. Of the wastes, 40% were found to be toxic to fathead minnows. Acute and embryo-larval toxicity of condenser water and its constituents from the Laramie Energy Technology Center Hanna-3 underground coal gasification condenser (UGC) were studied by DeGraeve *et al.*[356] and DeGraeve.[357] Separate acute tests with phenol, ammonia, and ammonia plus phenol showed that these constituents were the major components affecting acute toxicity to fish in the coal-conversion effluent. In addition, a toxicant avoidance system was developed and used to study the avoidance response of rainbow trout to phenol that comprised 26% of the total organic carbon in Hanna-3 condenser water. Wastewaters from a coal beneficiation plant were tested[358] for toxicity to channel catfish. In two of five 96-h static bioassays, mortalities occurred at high wastewater concentrations. Other tests using pH-altered reconstituted water with finely ground coal to simulate settleable solids caused mortality. Fish mortality was interrelated with settleable solids, pH 4.5, and low DO concentrations. Bergman *et al.*[359] and States *et al.*[360] conducted bioassays with coal processing wastewater on similar areas in the Great Plains. Rainbow trout were found to have varying sensitivity to effluents from solvent-refined coal and from *in situ* coal processing wastes. Levels varied from 10% to 90% effluent to achieve an LC_{50}. In a study of effects of leaching processing on Great Plains coal, Carlson *et al.*[361] found no severe adverse effects from acute and chronic toxicity testing on fathead minnows, rainbow trout, and bluegill sunfish (*Lepomis macrochirus*). Toxicity, growth retardation, and MFO induction were not observed in fish exposed to coal leachate, although a lowered spawning success rate was noted in fathead minnows. BCFs in the range of 1 000 to 5 000 were observed for several polynuclear aromatic hydrocarbons found in the leachates. The toxicity of distillery effluent to the cyprinid weed fish, *Rasbora daniconius*, was tested by Durve and Jain.[362] Concentrations of 1.5% of the effluent and higher caused mortality to fish and 50% of the fish were killed within 15 h when exposed to 3% of the effluent. Toxicity was found to be inversely proportional to pH but directly proportional to conductivity and alkalinity. The toxicity of an electroplating waste containing Cu, Cd, Zn, Pb, and heptachlor was examined by Shaffi.[363] Three-h exposures of nine species, *Cirrhina mrigala, Labeo rohita, Catla catla, Heteropneustes fossilis, Clarias batrachus, Mystus seenghala, Channa striatus, Channa punctatus*, and *Channa marulinus*, resulted in a lowering of glycogen levels and a raising of serum lactate levels. Most significant changes were between 10 to 15% effluent. Effects of exposure to effluent from a titanium dioxide industry located on the south-west coast of Finland were measured on perch (*Perca fluviatilis*) and bleak (*Alburnus alburnus*) by Lehtinen.[364] Both fish displayed significant decreases in the ability to compensate for torque in a rotating current when exposed to 300, 500, or 600 µl/l of the effluent for 28 d. A dose-dependent gill precipitate was thought to be the primary reason for this phenomenon. McBride *et al.*[365] exposed young-of-the-year rainbow trout to leachates from the Vancouver, B. C., sanitary landfill. They found that stored leachate exhibited a greater toxicity (6.5% versus 5.9% of the LC_{50}) than freshly collected material. Ammonia was the only chemical parameter tested that increased toxicity with storage. Haslouer[366] reported that there were 34 reported fish kills in Kansas in 1978 in which 76 000 fish died. Agricultural runoff and feedlot runoff were the most important cause of fish mortalities.

Pulp and Paper. The sensitivity of *Pimephales promelas, Daphnia magna*, and *Hydropsyche* sp. (caddis fly larvae) to various pulp and paper effluents was compared by Zanella and Berben.[367] The sensitivity of all species varied between materials and between plants of the same type. Bleached sulfite waste was the most toxic to *Daphnia magna*. Kruzynski[368] reported on the relationship of exposure of juvenile sockeye salmon (*Oncorhynchus nerka*) to dehydroabietic acid (DHA), a component of pulp and paper effluents, to various physiological parameters. A 120-h exposure in freshwater at a sublethal level of 0.65 mg DHA/l resulted in some mortality and much stress when the smolts were transferred to sea water. Oikari *et al.*[369] exposed caged fish in a kraft paper mill effluent stream. Rainbow trout and European perch were found to accumulate organic acids when exposed to such material. Histological changes in tissues of *Notoperus notoperus* induced by sublethal (10% by volume) and lethal (60% by volume) concentrations of pulp, paper, and mixed pulp and paper wastes were investigated by Verma *et al.*[370] Changes in the gill, liver, kidney, and intestine were greater in exposures to a sublethal than lethal concentration. The lethal concentration caused the greatest change in the gill compared to other organs. The tainting of rainbow trout exposed to kraft mill waste was reported by Liem and Naish.[371] An intermediate range

of 0.015 to 0.046% by volume caused a statistical difference between the apparent amount of tainting in these fish.

MISCELLANEOUS

The effects of strip mining on fish and diatoms in streams of the New River drainage basin in Tennessee were determined by Vaughan.[372] The trend for fish, especially *Semotilus atromaculatus*, and diatoms was toward smaller populations and fewer species as mining activity increased. Changes observed in the diatom community structure were thought to be related to differences in water quality as a result of increased runoff, sediment load, and siltation caused by mining activity. Egami and Hama[373] found that exposure of embryos of *Oryzias latipes* to more than 60 R/d for 2 months resulted in complete loss of reproduction capacity. However, secondary sex characteristics did develop in fish exposed to this concentration. Excretions of ^{131}I by catfish and bluegill (*Lepomis macrochirus*) were measured by Lyon *et al.*[374] Both species excreted ^{131}I at approximately the same rate although the bluegill had a higher initial level (213 counts/min bluegill versus 81 counts/min catfish). Shrivastava and Dwivedi[375] exposed fertilized eggs of the carp to high levels (100 to 5 000 R) of γ-rays. Hatchability was not affected but mortality was decreased by high doses and deformities and retarded development were noted. Brenniman *et al.*[376] found that relatively high levels of exposure of goldfish to outboard motor exhaust emissions produced undesirable effects, including mortality, changes in blood gases and pH, gross lesions of the coelomic cavity, skin, gills, and digestive system, and microscopic lesions of gills, liver, and kidneys. At least one aromatic hydrocarbon, toluene, was metabolized in a manner similar to that in mammals. The chronic effects of outboard motor exhaust products on fish physiology after exposure to more realistic concentrations found in rivers and lakes were not determined.

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